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TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

VOLUME XVIII.

CONTAINING THE REPORT OF THE PROCEEDINGS OF THE SOCIETY FROM OCTOBER, 1895, TO JUNE, 1897.

EDITED BY

WILLIAM S. CARTER, M.D.,

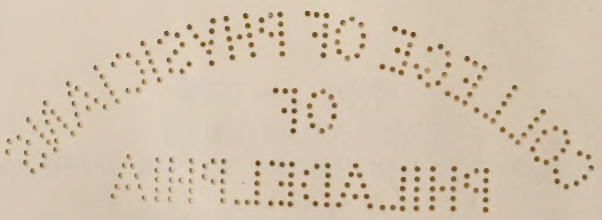
RECORDER OF THE SOCIETY.

NOV 3- 1898

PHILADELPHIA:
PRINTED FOR THE SOCIETY BY WM. J. DORNAN.
1898.

NOTICE.

THIS is the last bound volume issued by the Pathological Society. Since October, 1897, the reports of the proceedings have been published monthly, under the title of THE PROCEEDINGS OF THE PATHOLOGICAL SOCIETY OF PHILADELPHIA. Volume I. of the new series, covering the meetings of the Society from October, 1897, to June, 1898, has already appeared.



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FORMER PRESIDENTS.

SAMUEL D. GROSS, M.D., LL D., D.C.L. OXON., LL.D. CANTAB., elected 1857.

RENÉ LA ROCHE, M.D., elected 1858.

ALFRED STILLÉ, M.D , LLD., elected 1859, '61, and '62.

EDWARD HARTSHORNE, M.D., elected 1860 and '63.

J. M. DA COSTA, M.D., LL.D., elected 1864, '65 and '66.

JOHN H. PACKARD, M.D., elected 1867 and '68.

S. WEIR MITCHELL, M.D., LL.D., elected 1869.

JOHN ASHHURST, JR., M.D., LL.D., elected 1870.

JAMES H. HUTCHINSON, M.D., elected 1871 and '72.

WILLIAM PEPPER, M.D., LL.D., elected 1873.

H. LENOX HODGE, M.D., elected 1876.

S. W. GROSS, M.D , elected 1879.

JAMES TYSON, M.D., elected 1882 and '83.

E. O. SHAKESPEARE, M.D., elected 1884.

J. C. WILSON, M.D., elected 1885 and '86.

F. P. HENRY, M.D., elected 1887 and '88.

HENRY F. FORMAD, M.D., elected 1889 and '90.

ARTHUR V. MEIGS, M.D., elected 1891 and '92.

J. H. MUSSER, M.D., elected 1893, '94, '95, and '96.

OFFICERS AND COMMITTEES

OF THE

PATHOLOGICAL SOCIETY OF PHILADELPHIA.

(Elected at the annual meeting, October 10, 1895.)

President.

J. H. MUSSER, M.D.

Vice-Presidents.

J. GUITÉRAS, M.D., F. A. PACKARD, M.D.,
W. E. HUGHES, M.D., L. WOLFF, M.D.,

Secretary.

A. A. ESHNER, M.D.

Treasurer.

T. S. WESTCOTT, M.D.

Recorder.

W. S. CARTER, M.D.

Curator.

D. RIESMAN, M.D.

Business Committee.

A. C. ABBOTT, M.D., W. S. CARTER, M.D.,
C. W. BURR, M.D., A. HAND, Jr., M.D.,
J. A. SCOTT, M.D.

Committee on Publication.

H. W. CATTELL, M.D, F. P. HENRY, M.D.,
J. DALAND, M.D., F. A. PACKARD, M.D.

Committee on Morbid Growths.

J. McFARLAND, M.D., D. RIESMAN, M.D.,
A. STENGEL, M.D.

OFFICERS AND COMMITTEES

OF THE

PATHOLOGICAL SOCIETY OF PHILADELPHIA.

(Elected at the annual meeting, October 8, 1896.)

President.

J. H. MUSSER, M.D.,

Vice-Presidents.

W. E. HUGHES, M.D., F. A. PACKARD, M.D.,
C. W. BURR, M.D., J. GUITÉRAS, M.D.

Secretary.

A. A. ESHNER, M.D.

Treasurer.

T. S. WESTCOTT, M.D.

Recorder.

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D. RIESMAN, M.D.

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J. A. SCOTT, M.D.

Committee on Publication.

A. STENGEL, M.D. J. DALAND, M.D.,
H. W. CATTELL, M.D., F. P. HENRY, M.D.

Committee on Morbid Growths.

J. McFARLAND, M.D., A. O. J. KELLY, M.D.,
D. RIESMAN, M.D.

LIST OF MEMBERS.

Members marked O. M. are those originating the Society.

ELECTED.

1894 Abbott, A. C., 221 South Forty-fourth Street.
1887 Allyn, Herman B., S. E. corner Forty-second Street and Baltimore Avenue.
1889 Anders, J. M., 1603 Walnut Street.
1894 Arnold, John P., 3722 Walnut Street.
1861 Ashhurst, John, Jr., 2000 DeLancey Place.
1891 Ashton, Thomas G., 128 South Seventeenth Street.
1895 Ashton, William E., 2011 Walnut Street.

1897 Babcock, W. Wayne, 2133 North Front Street.
1898 Baggs, Albert N., Abingdon, Pa.
1887 Baker, G. F., 1818 Spruce Street.
1888 Balliet, Tilghman M., 3705 Powelton Avenue.
1885 Beates, Henry, Jr., 1504 Walnut Street.
1871 Bennett, Wm. H., 2105 Spruce Street.
1887 Berens, Bernard, 2002 Chestnut Street.
1894 Bevan, D., 1015 South Third Street.
1894 Beyea, H. D., 237 South Thirteenth Street.
1892 Birney, D. B., 1810 DeLancey Place.
1893 Bochroch, Max H., 968 North Eighth Street.
1886 Bodamer, George A., 1507 Girard Avenue.
1892 Boger, John A., 2213 North Broad Street.
1896 Booth, Arthur W., Episcopal Hospital.
1898 Boston, L. Napoleon, 1524 Snyder Avenue.
1893 Boyer, H. P., 1338 Spruce Street.
1857 Brinton, John H., 1423 Spruce Street. (O. M.)
1898 Brinton, Ward, 1423 Spruce Street.
1896 Brown, H. MacV., 915 South Forty-ninth Street.
1895 Brubaker, A. P., 105 North Thirty-fourth Street.
1893 Bryan, J. Roberts, 4200 Chestnut Street.
1887 Burr, Charles W., 1327 Spruce Street.

ELECTED

1893 Carpenter, H. B., 1523 Locust Street.
1897 Carpenter, J. T. Jr., 1419 Walnut Street.
1887 Cattell, H. W., 3709 Spruce Street.
1898 Chestnut, J. E., 1757 Frankford Avenue.
1887 Chrystie, Walter, Bryn Mawr, Pennsylvania.
1866 Cleemann, Richard A., 2135 Spruce Street.
1898 Coates, George M., Devon, Pa.
1872 Cohen, J. Solis, 1431 Walnut Street.
1883 Cohen, S. Solis, 219 South Seventeenth Street.
1896 Coley, Thomas Luther, 1339 Pine Street.
1890 Coplin, W. M. L., 1419 South Broad Street.
1898 Currie, Thos. R., 113 East Cumberland Street.
1871 Curtin, Roland G., 22 South Eighteenth Street.

1894 Da Costa, J. Chalmers, 1629 Locust Street.
1857 DaCosta, J. M., 1700 Walnut Street. (O. M.)
1885 Daland, Judson, 319 South Eighteenth Street.
1857 Darrach, James, 5020 Green Street, Germantown. (O. M.)
1890 Davis, C. N., 1931 Spruce Street.
1882 Davis, G. G., 255 South Sixteenth Street.
1893 Davisson, Alexander H., 2024 Pine Street.
1889 Deaver, H. C., 1534 North Fifteenth Street.
1885 Deaver, J. B., 1634 Walnut Street.
1882 Dercum, F. X., 1719 Walnut Street.
1895 Donnellan, P. S., 1028 Spruce Street.
1894 Dougherty, S. W., 215 South Seventeenth Street.
1888 Downes, Andrew J., 1725 Girard Avenue.
1889 Downs, Norton, 102 West Walnut Lane, Germantown.
1877 Drysdale, T. M., 1305 Locust Street.
1866 Duer, Edward L., 1606 Locust Street.
1876 Dulles, C. W., 4101 Walnut Street.
1883 Dunmire, G. B., 1618 Spruce Street.
1860 Dunton, W. R., High Street and Germantown Avenue, Germantown.

1896 Edsall, David L., 330 South Sixteenth Street.
1891 Eshner, A. A., 224 South Sixteenth Street.

1893 Farr, W. W., 5800 Green Street, Germantown.
1892 Fenn, William B., Denver, Col.

ELECTED

1881 Fenton, T. H., 1319 Spruce Street.
1857 Fischer, Emil, 729 North Sixth Street. (O. M.)
1876 Fisher, Henry M., 317 South Twelfth Street.
1894 Fox, L. Webster, 1304 Walnut Street.
1886 Freeman, W. J., 1720 Locust Street.
1889 Frese, Carl, German Hospital.
1895 Frazier, Charles Harrison, 133 South Eighteenth Street.
1880 French, M. S., 1437 Spruce Street.
1894 French, Samuel, Plymouth, Pa.
1890 Friebis, George, 1906 Chestnut Street.
1896 Fritz, Clarence H., 3932 Brown Street.
1885 Fussell, M. H., 189 Green Lane, Manayunk.

1895 Garitee, C. J., 1117 Spruce Street.
1897 Gerson, T. Perceval, Philadelphia Hospital.
1870 Getchell, Frank H., 1432 Spruce Street.
1894 Girvin, John H., 3912 Walnut Street.
1898 Gittings, J. Claxton, 4013 Chestnut Street.
1890 Gleason, E. Baldwin, 41 South Nineteenth Street.
1882 Godey, H. E., N. E. corner Nineteenth and Spruce Streets.
1886 Goodell, W. Constantine, 2041 Locust Street.
1890 Grayson, C. P., 251 South Sixteenth Street.
1867 Grier, Matthew J., 1531 Spruce Street.
1883 Griffith, J. P. C., 123 South Eighteenth Street.
1897 Gross, Wm., 701 North Fortieth Street.
1894 Grove, J. H., 1504 Arch Street.
1874 Guitéras, John, 130 South Thirty-ninth Street.
1890 Gummey, Frank P., 4774 Main Street, Germantown.

1896 Haden, H. C., 346 South Fifteenth Street.
1893 Hamill, S. M., 1822 Spruce Street.
1893 Hand, Alfred Jr., 211 South Seventeenth Street.
1885 Hare, Hobart A., 222 South Fifteenth Street.
1897 Harris, A. L., Jefferson Medical College Hospital.
1898 Hart, Chas. D., Pennsylvania Hospital.
1890 Hartzell, M. B., 3634 Chestnut Street.
1873 Hearn, W. Joseph, 1120 Walnut Street.
1870 Henry, Frederick P., 1635 Locust Street.
1896 Henry, John N., 1635 Locust Street.
1880 Hewson, Addinell, Jr., 1508 Pine Street.

ELECTED

1894 Hiller, Hiram M., 350 South Sixteenth Street.
1894 Hughes, George M., 241 North Eighteenth Street.
1882 Hughes, W. E., 3726 Baring Street.

1890 Izard, William, Sixth and Penn Streets, Camden, N. J.

1890 Jameson, W. B., S. E. corner Fortieth and Brown Streets.
1890 Jarrett, Henry, Camden, N. J.
1895 Johnston, John A., National Soldiers' Home, Virginia.
1895 Jopson, J. H., 334 South Sixteenth Street.
1898 Judson, Chas. F., 1934 Pine Street.
1885 Jurist, L., 916 North Broad Street.

1898 Kalteyer, F. J., 1338 Spruce Street.
1895 Kelly, A. O. J., 1911 Pine Street.
1890 Ketcham, S. Rush, 1708 Green Street.
1897 King, W. H., 412 South Fifteenth Street.
1888 Kirby, Ellwood, 1202 Spruce Street.
1894 Kirkbride, Thomas S., Jr., 1406 Spruce Street.
1897 Klein, Alexander, 513 Pine Street.
1890 Kneass, Samuel S., University Dormitories.
1895 Krusen, Wilmer, 158 North Twentieth Street.
1892 Kyle, D. Braden, 1517 Walnut Street.

1893 Lainé, D. T., 129 South Fifteenth Street.
1890 Laplace, Ernest, 1828 South Rittenhouse Square.
1898 Latta, Samuel W., 3626 Baring Street.
1894 Leach, W. W., 2118 Spruce Street.
1869 Leaman, Henry, 828 North Broad Street.
1888 Leidy, Joseph, 1319 Locust Street.
1887 Leopold, Isaac, 1520 Franklin Street.
1868 Lewis, Francis W., 2016 Spruce Street.
1875 Lewis, Morris J., 1316 Locust Street.
1894 Lincoln, Clarence W., Wayne Avenue and Coulter Street, Germantown.
1893 Lindsay, John, 340 South Fifteenth Street.
1886 Lloyd, James Hendrie, 3910 Walnut Street.
1898 Ludholtz, Edward, 3103 Diamond Street.
1894 Loeb, Ludwig, 1337 North Seventh Street.
1886 Longaker, D., 652 North Eighth Street.
1875 Longstreth, Morris, 1416 Spruce Street.

ELECTED

1880 McClellan, George, 1352 Spruce Street.
1892 McFarland, J., 421 West Price Street, Germantown.
1894 McKee, James H., 1519 Poplar Street.
1890 McKelway, George I., 1612 Locust Street.
1894 McLeod, George, 261 South Fifteenth Street.
1898 McReynolds, R. P., 3722 Walnut Street.
1887 Martin, Edward, 415 South Fifteenth Street.
1892 Massey, Frank, 3604 Locust Street.
1864 Mears, J. Ewing, 1429 Walnut Street.
1873 Meigs, Arthur V., 1322 Walnut Street.
1878 Mills, C. K., 1909 Chestnut Street.
1884 Mitchell, J. K., 256 South Fifteenth Street.
1896 Modell, D. A., 242 Fairmount Avenue.
1857 Morehouse, George R., 2033 Walnut Street. (O. M.)
1898 Morris, Henry, 315 South Sixteenth Street.
1885 Morrison, William H., Holmesburg, Pennsylvania.
1887 Morton, S. W., 113 South Twentieth Street.
1890 Muehleck, G. A., 1320 South Fifth Street.
1869 Müller, August F., 4753 Green Street, Germantown.
1880 Musser, J. H., 1927 Chestnut Street.

1879 Neff, J. S., S. W. corner Twenty-third and Spruce Streets.
1894 Newcomet, W. S., 3229 Powelton Avenue.
1898 Newton, R. D., 2705 Oxford Street.
1861 Norris, William F., 1530 Locust Street.

1894 O'Malley, James, 1104 Jackson Street.
1897 Ostheimer, Maurice, 1229 Locust Street.

1885 Packard, F. A., 110 South Eighteenth Street.
1893 Packard, F. R., 324 South Nineteenth Street.
1857 Packard, John H., Hotel Stenton. (O. M.)
1892 Pearce, F. S., 1407 Locust Street.
1896 Pease, H. D., 5812 Morris Street, Germantown.
1890 Pennock, W. J., 1407 North Seventeenth Street.
1891 Penrose, Charles B., 1331 Spruce Street.
1865 Pepper, William, 1811 Spruce Street.
1895 Perkins, F. M., 1428 Pine Street.
1897 Peters, L. C., 2136 Oxford Street.
1885 Piersol, G. A., 4722 Chester Avenue.

ELECTED

1894 Pitfield, Robert L., 4788 Main Street, Germantown.
1893 Posey, W. C., 1831 Chestnut Street.
1890 Potts, C. S., 1629 Locust Street.
1889 Price, Joseph, 241 North Eighteenth Street.

1885 Randall, B. A., 1806 Chestnut Street.
1898 Ravenel, M. P., University of Pennsylvania, Veterinary Department.
1894 Reckefus, C. H., 506 North Sixth Street.
1885 Reed, Boardman, 1831 Chestnut Street.
1894 Rhein, J. H. W., 1330 Spruce Street.
1897 Rhoads, Thomas Leidy, 1703 Walnut Street.
1894 Riesman, David, 326 South Sixteenth Street.
1891 Ring, G. O., 1442 North Thirteenth Street.
1876 Roberts, John B., 1627 Walnut Street.
1884 Robertson, W. E., 912 North Fourth Street.
1889 Robinson, William Duffield, 2012 Mount Vernon Street.
1891 Rosenthal, E., 517 Pine Street.
1894 Ross, George, 637 North Sixteenth Street.

1896 Sailer, Joseph, 330 South Sixteenth Street.
1867 Santee, Eugene J., 605 North Eleventh Street.
1890 Sartain, Paul J., 212 West Logan Square.
1895 Schamberg, J. F., 831 North Broad Street.
1897 Schreiner, E. R., N. E. corner Thirty-eighth and Spring Garden Streets.
1882 de Schweinitz, G. E., 1401 Locust Street.
1890 Scott, J. A., 121 South Eighteenth Street.
1898 Sharpless, Wm. T., West Chester, Pa.
1885 Shober, J. B., 112 South Seventeenth Street.
1889 Shoemaker, Harvey, 109 South Twentieth Street.
1868 Sinkler, Wharton, 1606 Walnut Street.
1881 Skillern, P. G., 241 South Thirteenth Street.
1881 Skillern, S. R., 3509 Baring Street.
1898 Smyth, Henry Field, Germantown Hospital.
1894 Spellissy, Joseph, 108 South Eighteenth Street.
1896 Spiller, William G., 4409 Pine Street.
1890 Stahl, B. F., 1502 Arch Street.
1894 Steele, J. D., 4504 Chester Avenue.
1889 Stengel, Alfred, 1811 Spruce Street.

ELECTED

1889 Stevens, Arthur A., 314 South Sixteenth Street.
1886 Stewart, David D., 108 South Seventeenth Street.
1896 Stewart, Alonzo H., 250 North Twelfth Street.
1857 Stillé, Alfred, 3900 Spruce Street. (O. M.)
1884 Stritmatter, I. P., 999 North Sixth Street.
1869 Stryker, Samuel S., 3821 Walnut Street.
1898 Stubbs, R. P., Presbyterian Hospital.
1894 Swan, John M., 807 North Forty-first Street.

1895 Taylor, A. E., 334 South Sixteenth Street.
1896 Taylor, J. Gurney, Pennsylvania Hospital.
1897 Teller, Wm. H., 1934 Green Street.
1894 Thomas, W. Hersey, 1445 North Seventeenth Street.
1894 Thomson, Archibald G., 1426 Walnut Street.
1868 Thomson, William, 1426 Walnut Street.
1897 Tucker, Henry, 19 South Twenty-first Street.
1892 Tunis, J. P., 129 South Eighteenth Street.
1898 Twaddell, Thomas, Cartwright, Elk Co., Pa.
1863 Tyson, James, 1506 Spruce Street.
1890 Tyson, T. Mellor, 1506 Spruce Street.

1895 Vanderwoort, C. A., 3250 North Fifteenth Street.
1887 Vansant, Eugene L., 1929 Chestnut Street.

1898 Wadsworth, W. S., Presbyterian Hospital.
1891 Wallace, James, 350 South Sixteenth Street.
1890 Warder, Charles B., 1401 North Sixteenth Street.
1874 Warder, W. H., 1212 North Broad Street.
1895 Wendell, W. G., 4126 Chester Avenue.
1887 Westcott, T. S., 108 North Nineteenth Street.
1885 Wharton, H. R., 1725 Spruce Street.
1897 White, Courtland Y., 1808 Diamond Street.
1893 White, F., Elwyn, Pennsylvania.
1873 White, J. William, 1810 South Rittenhouse Square.
1893 Whiting, A. D., 1523 Spruce Street.
1868 Willard, De Forest, 1601 Walnut Street.
1897 Williams, Henry L., 112 South Eighteenth Street.
1898 Willson, R. N., Pennsylvania Hospital.
1869 Wilson, James C., 1437 Walnut Street.
1891 Wilson, Samuel M., 1517 Arch Street.

ELECTED

1891 Wilson, W. R., 112 South Twentieth Street.
1895 Witmer, A. F., 322 South Fifteenth Street.
1898 Woldert, E. A., 1709 North Sixteenth Street.
1890 Wolff, L., 333 South Twelfth Street.
1890 Wood, A. C., 1501 Walnut Street.
1898 Wood, George B., 1925 Chestnut Street.
1865 Woods, D. Flavel, 1501 Spruce Street.
1897 Woodward, E. B., 4222 Chestnut Street.

1897 Yarrow, Thomas J., Jr., Episcopal Hospital.

1892 Ziegler, Jacob L., Mt. Joy, Lancaster County, Pa.
1895 Zimmerman, Mason W., 1622 Locust Street.

NON-RESIDENT MEMBERS.

Ashhurst, W. W., Chihuahua, Mexico.
Baines, A. S., Jr., 4484 Finney Avenue, St. Louis, Mo.
Ball, M. V., Warren, Pa.
Breinig, P. B., Bethlehem, Pa.
Brooke, B., Radnor, Pa.
Carter, W. S., Galveston, Texas.
Cerna, D., Galveston, Texas.
Da Costa, J. C., Jr.
Dock, George, Ann Arbor, Mich.
Edwards, W. A., San Diego, Cal.
Egbert, Joseph P., Wayne, Delaware County, Pa.
Eskridge, J. T., Denver, Col.
Fox, J. M., Torresdale, Pa.
Gaylord, H. R., Buffalo, N. Y.
Hamaker, W D., Meadville, Pa.
Hamann, C. A., 517 Euclid Avenue, Cleveland, Ohio.
Harrison, W. H., Harrisburg, Pa.
Hatch, John L., 2010 Fifth Avenue, New York City.
Hileman, J. B., Altoona, Pa.
Hunt, J. R.
Jamar, John H., Elkton, Md.
Lincoln, W. R., Cleveland, O.
McCarthy, D. J.
Muhlenberg, Frank, Reading, Pa.
Nancrede, C. B., Ann Arbor, Mich.
Osler, William, Johns Hopkins Hospital, Baltimore, Md.
Pfouts, G. B., Morris Plains, N. J.
Powell, W. M., Atlantic City, N. J.
Rahter, C. A., Harrisburg, Pa.
Shumway, E. A.
Slifer, Henry F., North Wales, Pa.
Smith, Allen J., Galveston, Texas.

Stiles, G. A., Conshohocken, Pa.
Taylor, L. H., Wilkesbarre, Pa.
Toulmin, H., Milwaukee, Wis.
Wells, G. M., Wayne, Delaware County, Pa.
Wetherill, R. B., Lafayette, Ind.
Wilmarth, A. W., State Home for the Feeble-minded, Chippewa Falls, Wisconsin.

CORRESPONDING MEMBERS.

ELECTED

1866 Bumstead, Freeman J. Died 1879.
1859 Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York. Dead.
1898 Cornil, V., Faculté de Médecine, Paris, France.
1859 Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York. Dead.
1885 Dent, Clinton T., Assistant Surgeon and Lecturer on Practical Surgery at St. George's Hospital, Surgeon to Belgrave Hospital for Children, London, England.
1860 Ellis, Calvin. Died 1883.
1888 Fedeli, Gregorio, Rome, Italy.
1858 Flint, Austin, Sr. Died 1886.
1890 Gibbes, Heneage, Ann Arbor, Michigan.
1858 Hammond, William A., Member of the National Academy of Sciences, New York.
1860 Isaacs, Charles E., Brooklyn. Died June 16, 1860.
1878 Jackson, J. B. S., Boston. Died 1879.
1886 Pye-Smith, P. H., Physician and Lecturer on Medicine at Guy's Hospital, London, England.
1860 Reeves, James E., Wheeling, West Virginia. Dead.
1861 Rokitansky, Carl. Died 1878.
1898 Virchow, Rudolf, Berlin, Germany.
1860 Watson, John, New York. Died June 3, 1869.
1898 Welch, Wm. H., Professor of Pathology, Johns Hopkins University, Baltimore.
1859 Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

REPORT.

I. THE OSSEOUS SYSTEM.

1. *Tuberculosis of metacarpus and metatarsus.*

Exhibited by Herman B. Allyn, M.D.

The patient from whom these specimens were obtained was a girl aged fourteen years. Her maternal grandfather had tuberculosis, but no tuberculosis exists in father, mother, and two brothers, who are living and well.

As a child, the patient suffered with rheumatic pains in the legs, which were so severe as often to compel her to stop and sit down when out for a walk. In appearance she was a plump anemic, but color came readily under excitement. For the past three years I have thought her distinctly anemic. Almost from infancy her appetite has been poor and capricious, the child living principally on milk. Meats she scarcely ever touched, and oysters never. The smell of fish was nauseating. Rice she disliked. She enjoyed pastries, sweets, and delicacies, but never ate immoderately of them. The bowels were costive; she was rather languid and emotional, and easily tired. She seemed to require more than the average amount of sleep, was easily chilled, and needed considerable bedclothing to keep her warm.

In June, 1893, she developed a swelling of the head of the first left metacarpal bone. This was pale, a little tender and painful, and somewhat warmer than the other. It subsided gradually, but not completely. There were, however, no persistent pain, tenderness and impaired motion. In damp, cold weather the discomfort would be greater; on the other hand, it would improve in dry, warm weather.

In the summer of 1894 she developed a prominent, cushiony swelling over the left wrist. It was accompanied by stiffness, pain, and tenderness; but there was very little heat. In the early fall of 1894 she began to complain of stiffness, pain, and swelling of the left ankle and instep, so that she had considerable difficulty in buttoning her shoe and in walking. After walking a while the stiffness became less, and in the afternoons she would be fairly comfortable. The difficulty with the foot was at

times much greater than at others. There was no history of injury of any kind, and neither cut nor puncture nor bruise preceded the swellings. By December, 1894, the swellings of wrist and foot became worse; she had fever, and was manifestly ill. Up to this time she had had no treatment.

Gradually a distinct arthritis of the left wrist and left metacarpophalangeal joint developed. In a short time a puffy, elastic swelling appeared over the right metatarsus. At times there was also pain, with stiffness, in the right wrist, and a gelatinous exudate formed in the right prepatellar bursa, but without pain or tenderness. The fever was irregular, but at first slight. Salicylate of soda seemed to have a decided influence on the pain and swelling, but when given in adequate doses it caused complete anorexia with nausea, and therefore had to be discontinued.

By the latter part of March the most prominent part of the left ankle began discharging an odorless, yellow, serous fluid. The same thing subsequently occurred with the left wrist. After the yellow, straw-colored, odorless discharge had continued for five or six weeks, it became purulent, and on April 18th Dr. Joseph McFarland found twelve tubercle bacilli in four cover-slip preparations. Gradually other openings over the carpus appeared, and the wrist-joint became disorganized, complete internal rotation occurring. The destructive process was not as great in the ankle.

The naturally poor appetite of the child made it impossible to provide sufficient nourishment. The discharging foci were horribly offensive, and toward the last emaciation became extraordinary. The lungs, heart, and kidneys remained apparently uninvolved. That is to say, they gave no demonstrable evidence of disease. Death occurred October 10, 1895.

October 24, 1895.

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2. *Tuberculosis of the hip-joint.*

A Verbal Communication by John Ashhurst, Jr., M.D., LL.D., with Description of Specimens by Joseph P. Tunis, M.D.

When your President asked me to make a few remarks this evening on some subject in surgical pathology, after consultation with Dr. Tunis, who kindly consented to assist me, we thought it might be of interest to bring forward the subject of tuberculosis of the hip-joint, although the society had had, not very long ago, a meeting principally

devoted to the general subject of tuberculosis of the bones and articulations. In the remarks which I had the honor of making at that meeting I dwelt more upon the general subject of osseous tuberculosis, and this evening I propose to confine myself to the disease as affecting the hip-joint, which has not always received the attention it deserves. In the first place, I think we may classify cases of hip-disease into three varieties, the rheumatic, the simple traumatic, and the tuberculous. The two first named are sometimes spoken of as cases of arthritic coxalgia, which is not really a good name to apply in this connection, because all forms of coxalgia which involve the joint tissues as distinct from the bone may properly be called arthritic. There is a not infrequent form of coxalgia, or hip-disease, which is really a rheumatic synovitis. We have in these cases a history of the child being exposed to cold or wet, sitting down on a cold stone or on the wet grass, for example, and oftentimes symptoms of hip-disease will thereafter develop. These patients improve under careful treatment, and the child is ultimately as well as before. These might properly be called cases of rheumatic synovitis of the hip-joint, and the patient may be expected to recover as he would from a similar condition of any other part. Then there are cases in which, occasionally, a mild form of synovitis of the hip-joint seems to follow traumatism. These are not very common, because the hip-joint is so deeply placed as to escape in most instances; but we see cases in which children have met with doubtful injuries previous to the development of coxalgic symptoms. For instance, the parent thinks that the child may have fallen from its coach, or from a stool, to the ground, and while the history is very obscure, and while there are no external marks of injury, the child shortly develops symptoms pointing to hip-disease, which last for a variable period, eventually passing off and the child getting well. It is possible that there may have been in such cases a simple synovitis, the result of contusion of the hip-joint, just as we may have a synovitis, the result of contusion of the knee. The large majority of cases of hip-disease, however, have as their essential lesion the existence of tuberculosis in some of the tissues about the joint, and superadded in many cases to the previous existence of the tubercle-bacillus in some part of the body is a slight traumatism, which seems to determine the locality in which the disease shall be developed. Often the child may have had a number of slight falls, perhaps falls on the ice, or even in the house, and a child that is naturally awkward is, of course, more predisposed to fall than others. Again, boys, from the rougher plays in which they engage, fall more than girls, and we find that hip-disease is nearly twice as frequent in

the male as in the female. Hip-disease follows these falls, but the child previously has had tuberculosis in some part of its body. Modern pathologists believe that the tubercle bacilli are very widely distributed, and may be stored up, as it were, in some part of the body, probably in the bronchial glands. As the result of traumatism, what the Germans call a place of less resistance is developed, and then the tuberculous action begins. Thus we have in the case of a tuberculous child the history of a fall or a succession of slight falls, which establish a place of less resistance in some of the tissues connected with the hip-joint. The tubercle establishes itself at that point and goes on to the production of hip-disease. The traumatisms concerned in the production of hip-disease are usually slight. A person who has received a severe injury of the hip may have a fracture or a dislocation, but is not apt to become the subject of hip-disease. It is from a succession of slight

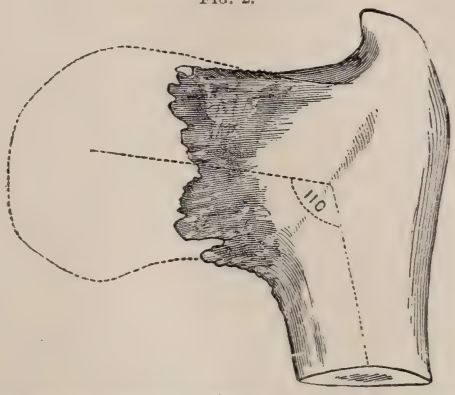
FIG. 1.

Complete loss of the head of the bone down to the anatomic neck. There is apparently no disease of the neck of the bone itself, and therefore no alteration in the normal angle between the neck and the shaft of the bone.

bruises rather than from any great violence that the condition is established which leads to this affection. The lesions of tuberculous hip-disease may begin in any one of the constituent parts of the joint. I do not believe that any portion of the joint is exempt in this respect. Tuberculosis may exist first in the head of the bone, constituting a form of hip-disease of which we have specimens here. (Figs. 1 and 2.) We have other cases in which the disease shows itself first in the neck of the bone (Fig. 3), the head being unaffected even at a very late period. The destruction of the neck sometimes leads to separation of the head (Fig. 4), which remains as a sequestrum in the articular cavity. Then there are other cases, though not very common, in which the disease begins in the acetabulum. A frequent classification of cases of hip-disease is into the arthritic (those affecting only the soft tissues), the

femoral, and the acetabular, and attempts have been made to differentiate between the latter by the position of the sinuses which form in

FIG. 2.



Complete loss of the head and extensive disease of the neck of the bone, associated with an alteration of the angle, reducing the latter to 110°. The dotted lines indicate the loss of substance, which has extended down below the surgical neck.

the progress of the disease. In cases of femoral coxalgia the sinuses are on the outer portion of the limb, whereas in acetabular coxalgia

FIG. 3.

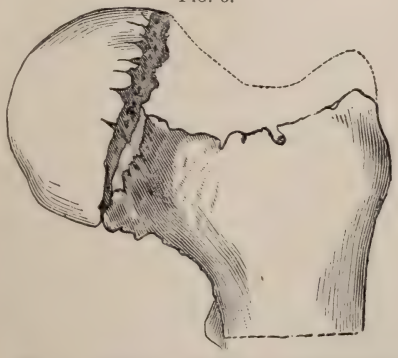


FIG. 4.

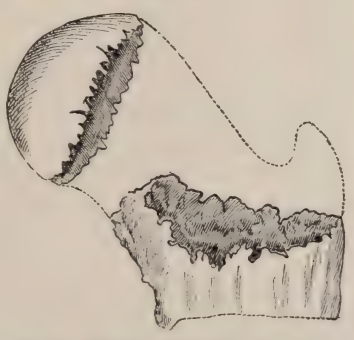


FIG. 3.—Results of rarefying osteitis, probably tuberculous, affecting principally the neck of the bone. The head of the femur is shown to be for the most part free from disease, and only that portion in the neighborhood of the anatomic neck has been affected. Marked shortening has occurred in this case, and great loss of bone-tissue as the result of the continued suppuration. The head of the bone was found firmly fixed in the acetabulum, and was removed separately.

FIG. 4.—Extensive disease of the neck of the bone from the anatomic neck to the surgical neck of the femur. The dotted lines represent diagrammatically the amount of tissue lost.

they are on the inner side, while in cases in which the acetabulum has been perforated they may open in the groin above Poupart's ligament.

These diagnostic marks, however, are not very satisfactory, because in advanced cases we usually have both parts involved. While I would not deny that the acetabulum may be primarily the seat of disease, it is certainly much less often so than the head or neck of the femur, as is shown by the fact that in most cases the acetabulum is only slightly implicated even when the femoral disease is far advanced. We may also have hip-disease arising in the soft tissues of the joint, not merely in the mild forms of synovitis which get well under simple treatment, but we may have tubercle making its appearance first in some of the soft tissues, and especially, I believe, in the ligamentum teres. The points to which I wish particularly to invite the attention of the society to-night are the function of the ligamentum teres as explaining the circumstance under which it becomes involved in hip-disease—and it is my belief that hip-disease begins here oftener than is usually supposed —and the very marked changes observed in the head and neck of the femur, and the explanations which may reasonably be given as to their occurrence.

Taking up the bone-changes first, we have in one case the head remaining and the neck partially absorbed, or perhaps disappearing completely, while in other cases we have the neck of the femur remaining, though the head of the bone may be reduced to a mere osseous button. The changes in the head of the bone may be due to several causes. Oftentimes tubercle is deposited in the first instance in the caput femoris, and it is easy to understand that with the extension of the disease the bone will become softened, and that portions may be thrown off into the joint as the result of the carious process, the head thus becoming gradually disintegrated. Then there is another cause to which attention has been called by Dr. Lovett, of Boston, who believes that the reason that the head of the bone disappears in some cases is its attrition against the acetabulum, gradually causing its disintegration. Dr. Lovett advocates the treatment of hip-disease by powerful traction, or what he calls *distraction*, and he gives one illustration showing the upper end of the femur in a patient who had been thus treated, and a second representing the specimen from a case of about equal severity, in which the patient had been treated without traction. In the first instance the head of the bone was but slightly worn away, while in the other case it had almost disappeared. I have no doubt that Dr. Lovett is correct in his views upon this subject. Then there are cases in which the articular cartilage being separated, the bone is exposed to prolonged maceration in the purulent contents of the joint, and becomes roughened and finally disappears, to a certain extent, as a

consequence of this maceration. We see the same thing in cases of empyema, where, the pus having been discharged through an incision, the rib sometimes becomes carious or necrosed by maceration, and partial disintegration follows. The same thing is seen in the secondary disease of the vertebræ which sometimes follows upon such an affection as perinephric abscess. The coexistence of vertebral caries and a lumbar abscess does not necessarily prove that the abscess is of spinal origin. We may have a perinephric abscess causing superficial caries of the vertebræ by prolonged contact of the bone with the pus of the abscess cavity, and similarly may have maceration of the head of the femur by pus in the hip-joint. The disappearance of the caput femoris in hip-disease may therefore be due to a combination of three causes: 1, the presence of tubercle; 2, the attrition of the head of the bone against the acetabulum; and, 3, the maceration of the bone in the purulent contents of the joint.

As regards the changes in the neck of the bone, we have a very interesting alteration in the shape of the neck and in the angle which it makes with the femur. This is very well shown in some of these specimens. The head of the bone in the normal position (Fig. 5) occupies a position considerably above the level of the trochanter, and the neck joins the shaft at an angle of about 140 degrees. This varies in the individual at different periods of life, but in the normal bone the neck always makes an obtuse angle with the shaft. Here is a specimen (Fig. 6) in which the disease appears to have affected principally the soft tissues, and yet there is a change in the bone, the neck being shortened and the angle of its attachment being reduced to about 130 degrees. In another specimen there has been extensive disease of the bone itself, and the head has separated from the neck, which occupies a position at right angles to the shaft. Here is another specimen (Fig. 2, *supra*), showing a case in which the head and neck of the bone have almost entirely disappeared, but what is left of the neck occupies a position at right angles to the shaft. This change in the neck of the bone is very striking, and is due to a form of osteitis which is often spoken of as rarefying osteitis, but which is not necessarily a tuberculous lesion. It is met with very often as the result of ordinary traumatism. It is a change, too, that we are familiar with in old persons, and is usually spoken of as a senile change. It has been believed that old age of itself leads to this change in the angle of the bone, and its occurrence is given as one of the reasons why old persons are more subject than younger ones to fracture of this part. But Sir George Humphry, of Cambridge, England, whom I regard as one of the very highest author-

ities on bone-pathology, has contravened this view, and has established the fact that this change of angle is not properly a senile change, but that it is a pressure-change. He maintains that the change is gradually brought about in the course of a long life by the weight

FIG. 5.

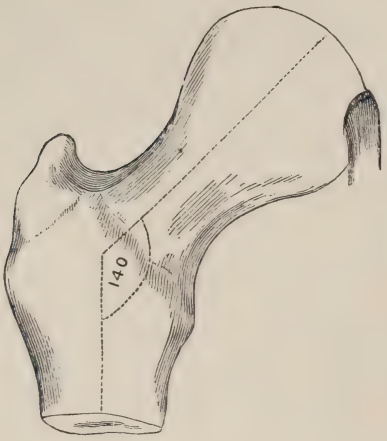


FIG. 6.

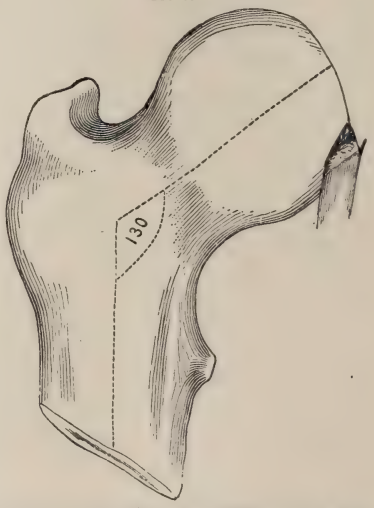


FIG. 5.—A perfectly healthy femur-head, removed post mortem from a child in whose case the opposite caput femoris had been removed by excision six years previously, with marked improvement resulting. The angle of the neck with the shaft is shown to be about 140°. The ligamentum teres is represented in its normal position.

FIG. 6.—Marked shortening of the neck of the femur and decrease of the angle between the neck and the shaft. The angle is reduced in this specimen to 130°. The position of the ligamentum teres is well shown. The deformity has been the result of a rarefying osteitis of the neck of the femur. The head of the bone is not diseased.

of the body being sustained upon the necks of the femurs, thus gradually pressing down the heads of the bones. It is therefore the result of pressure from walking through a long period of years, and is not the result of age. Sir George Humphry has demonstrated this by comparative observations in persons who had been subjected in early life to amputation of one of the lower limbs. In cases, for example, in which there had been an amputation of the right leg, so that it was necessarily relieved from the pressure caused by walking during the rest of the patient's life, the weight of the body being borne upon the left leg, he found that in the limb which had thus been relieved from pressure, although the person may have lived to extreme old age, the neck of the femur retained its normal angle, whereas in the limb which had been exposed to pressure, not having been amputated, and therefore

continuing to sustain the weight of the body, the so-called senile change was thoroughly established, and, in some instances, even more marked than usual. He has thus made it very clear that this change, which is ordinarily observed in advancing years, is not really dependent upon old age, but is simply a result of pressure. We may have a rarefying osteitis which, by softening the bone, hastens this change, and cases are on record in which this condition, ordinarily met with only in advancing years, has been produced in the course of a few months as the result of traumatism. There have been suits for malpractice under such circumstances, the accusation being made that the surgeon had not recognized a fracture of the neck of the bone, because when the patient was discharged from treatment the limb was found to be shortened. I believe it, however, to be a well-established fact that as the result of traumatism this conditon of rarefying osteitis may occur, and the so-called senile change be brought about in a short time as the result of pressure upon the softened bone. The same thing takes place in these cases of hip-disease. The bone becomes softened from the presence of tubercle, and then the angle of the neck changes as the result of pressure through the child walking upon the limb. The change, therefore, results from pressure on the softened bone, and not necessarily from the presence of tubercle or of osteitis, which would not produce the effect if pressure could be avoided. Clinically, we find many cases in which this change has occurred. The neck of the bone, instead of occupying its normal position at an obtuse angle with the shaft, makes with the latter a right angle, or even an acute angle. This is the chief cause of the shortening in the advanced stages of hip-disease. It has been supposed that in these cases the head of the bone was dislocated, and in some cases dislocation actually occurs. I have found, in the operation of excision, the head of the bone resting on the ilium above the acetabulum. But in most cases where dislocation is supposed to have occurred there is really no displacement of the head, but the angle of the neck of the bone is changed so that the trochanter slips as it were beyond the head, which still remains in its socket. This may occur in connection with caries, or with that condition which Sir James Paget calls interstitial absorption, where suppuration may be entirely absent. This interstitial absorption is frequently met with in the vertebræ, where it causes great deformity, and more rarely in the long bones. We have the same interstitial absorption occurring in hip-disease, and we see its effects in many cured cases, the patient getting well under careful treatment without any abscess forming, but with marked shortening due to the head and neck of the bone being partially absorbed, and to its angle being

changed. In other cases, of course, abscesses form in connection with ordinary caries, which is very often tuberculous, though not necessarily. The head of the bone may then be separated, and may be found completely loose in the joint. Disease of the acetabulum is comparatively rare, and I do not know that I have ever in excising the hip-joint found the acetabulum extensively involved while the femur had escaped. It is, on the other hand, very common to find the femur extensively and the acetabulum only slightly diseased. It is usually involved to a slight extent, but only in advanced cases of hip-disease is it much affected. It is seldom involved to anything like the same degree that the femur is, and in the acetabulum necrosis often takes the place of caries.

Disease of the hip may also begin in the soft tissues of the joint, particularly, as I have already said, in the ligamentum teres. Most surgeons are, I think, under the impression that this ligament has no function, and this view is encouraged by the opinion of many anatomists, who maintain that it is almost constantly in a state of relaxation. I cannot but believe, however, that the ligamentum teres really has an important function, and that it is in connection with this function that its liability to injury—and in consequence to become the seat of tubercle—occurs, just as we find, in other regions of the body, that parts which are functionally active are especially exposed to traumatism. The lower jaw, for example, is much more liable to fracture than the upper jaw, and the clavicle is much oftener injured than the scapula. Another common impression is that the ligamentum teres runs from about the center of the acetabulum directly across the joint to the head of the femur, but this is not correct. The drawings which Dr. Tunis has had placed on the blackboard, taken from Testut[1] (Figs. 9 and 10), illustrate what is really the normal position of the ligamentum teres. You will observe that it is a wide band attached to the lower edge of the acetabulum, divided below into two branches, and passing up in contact with the femur to which it is joined at a depression in the head of the bone. You will notice that when the patient is erect it is almost a vertical ligament. Now, I believe, and I think that this view is sustained by the evidence derived from a study of hip-disease, that, as long since pointed out by Turner and Savory,[2] the ligamentum teres really has an important function, and may be properly described as a suspensory ligament of the trunk. Of course, the whole weight of the body is not sustained by this ligament, but mostly by the head of the femur in more or less close contact with the cotyloid cavity; but this ligament,

[1] E. Testut, Traité d'Anatomie, tome i., pp. 435, 436. Deuxième edition. Paris. 1893.
[2] Journal of Anatomy and Physiology, vol. viii.

I believe, serves to diminish the pressure of the pelvis upon the head of the bone, and to lessen strain. The position of the ligamentum teres is like that of the old-fasioned leather springs which used to be found in stage-coaches, the body of the coach being balanced upon two broad strong bands of leather. The pelvis is to a certain extent similarly balanced upon these ligaments on either side, which are attached to the heads of the thigh-bones, and then pass down to the lower edges of the cotyloid cavities. I believe that their function is to ease up, as it were, and prevent sudden concussions, which would otherwise be experienced at every step that we take. If these ligaments were absent, the cases in which we have disease as the result of concussion or slight contusion of the hip-joint would be much more common than they are now. That the anatomic relations of the ligamentum teres are what I have described, is shown by these specimens, in which a portion of the liga-

FIG. 7. FIG. 8.

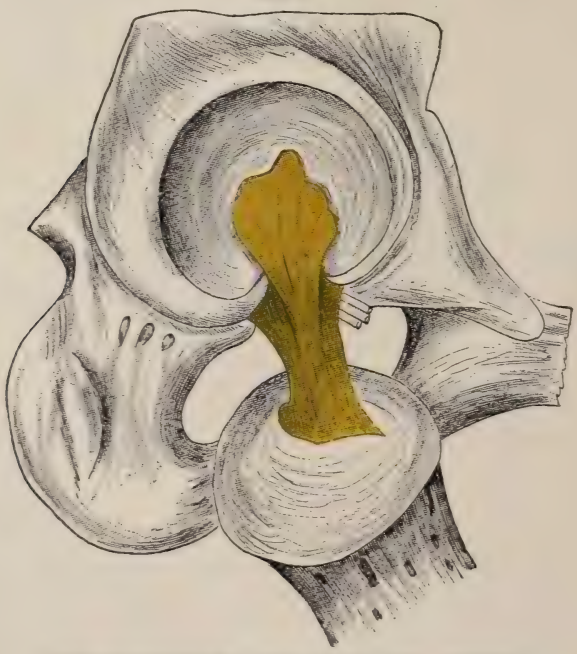
FIG. 7.—Anterior aspect of the head of a femur in which the articular cartilage was principally involved. The neck of the bone has been shortened, and the angle of the neck with the shaft reduced to 128°. The peeling off of the articular cartilage of the head of the femur and the erosion of the cancellous bone are well shown.

FIG. 8.—Posterior aspect of the same bone. Change of angle to 128°, shortening of the neck, and necrosis of the articular cartilage are well shown. There is also a pocket of diseased bone at the anatomic neck of the femur.

ment is still attached. (Figs. 5 and 6.) Here is one where the neck of the bone is almost entirely gone, while the head is hardly involved at all. The ligamentum teres is seen passing downward, hugging, as it were, the head of the bone, until it reaches its point of attachment at the lower part of the acetabulum. As a result of the stress put on the ligament in the various movements of the part it becomes weakened,

and thus a place of less resistance; a deposit of tubercle follows; and the ligament, softened and partially disintegrated, ceases to protect the joint, the other constituents of which then suffer from the effects of concussion and other slight traumatisms, and in turn become tuberculous, this primary lesion of the ligamentum teres thus furnishing a prolific cause of hip-disease. The older writers maintained the importance of the ligamentum teres in the development of hip-disease, and I think with reason. I believe, then, that we may have tuberculosis affecting the head and neck of the femur primarily and the acetabulum secondarily; or, more rarely, the acetabulum primarily and the other parts

FIG. 9.



Origin and termination of ligamentum teres. (POIRIER.)

secondarily. In other cases the disease affects first the soft tissues, and particularly the ligamentum teres. In some cases the neck of the bone is shortened and its angle changed, or it may be so much disintegrated that the head becomes separated while itself but little affected. In other cases the head of the bone may have almost disappeared, and what is left of it may yet be attached to a nearly unchanged neck. In still other cases the disease, beginning in the ligamentum teres, the soft tissues

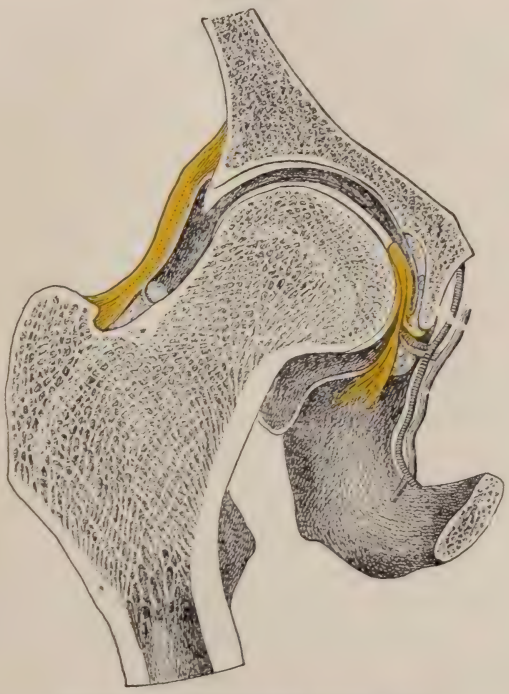
of the joint may be extensively diseased and the caput femoris deprived of its articular cartilage, while in other respects the bone is but little affected. (Figs. 7 and 8.)

Dr. Tunis has had drawings made from these specimens, illustrating the different points to which I have asked your attention, and these he will now present for your examination.

Description of the Specimens.

Before taking up in detail the interesting series of specimens which have been selected from a large number of femoral heads removed by

FIG. 10.



Position of ligamentum teres in erect posture.

excision at different times at the Children's Hospital, it may be well to review, briefly, some of the more important points of the anatomy of the hip-joint. (See Figs. 9 and 10).

In addition to the muscles which surround and give support to the head of the femur at the point where it is received into the os innominatum, there are a number of ligaments peculiar to this joint—namely,

the iliofemoral, the ligamentum teres, the cotyloid, and the transverse. The cotyloid ligament occupies the brim of the acetabulum and helps to deepen that cavity. A reference to the accompanying illustration (Fig. 10) will show the relative position of the component parts of this joint. We wish especially to call attention to the ligamentum teres.

In Morris's *Anatomy*[1] its origin and insertion are most carefully gone into, and six different plates used to illustrate the manner by which it may be put upon the stretch. "It is an intra-articular flat band which extends from the acetabular notch to the head of the femur, and is usually about an inch and a half long. It has two bony attachments, one on either side of the cotyloid notch immediately below the articular cartilage, while intermediate fibers spring from the under surface of the transverse ligament. The ischial portion is the stronger, and has several of its fibers arising outside of the cavity, below, and in connection with the transverse ligament, where it is also continuous with the capsule and periosteum of the ischium. At the femur it is fixed to the front part of the depression on the head, and also to the cartilage round the margins of the depression."

"The ligamentum teres[2] is of but very little use in resisting violence or giving strength to the joint. It assists in checking rotation outward and abduction during flexion. A ligament can only be of use when it is tight, and it was found by trephining the bottom of the acetabulum, removing the fat, and threading a piece of whip-cord round the ligament, that the ligament was slack in simple flexion, and very slack in complete extension, but that its most slack condition was in abduction. It is tightest in flexion combined with abduction and rotation outward, and almost as tight in flexion with outward rotation alone, and in flexion with abduction alone."

It is also of interest as bearing practically on the subject, that the angle which is made by a line drawn through the axis of the neck meeting a line drawn through the axis of the shaft of a bone ranges between 110° and 140°. It is a smaller angle in women and children, according to Quain's *Anatomy*.[3]

Although it is a commonly accepted idea that the ligamentum teres is an unimportant ligament of the hip-joint, it seems more rational to believe that it is of considerable importance, especially when we consider its topographic anatomy. While it is usually believed that the ligamen-

[1] Morris's Anatomy, p. 266, ed. 1893, P. Blakiston, Son & Co., Phila.

[2] Ibid., p. 270.

[3] Quain's Anatomy, Part I., vol. ii., 1890, p. 123. See also Humphry's Journal of Anatomy vol. xxiii., p. 273; and W. Braune's monograph in German.

tum teres stretches from the cotyloid notch directly over to the head of the femur, such is not really the case; but the ligament, on the contrary, passes from the depression in the head of the femur to the lower border of the cotyloid notch, as is well shown in Fig. 10. In this way, instead of being an unimportant ligament, the ligamentum teres plays an active part in the mechanism of the hip-joint, helping, as it were, to suspend the pelvis and the organs contained within and above that bony arch on a framework of which the shafts of the femurs act as the principal support. Examining into the function of the ligamentum teres from this point of view, it would at once appear that it assists in preventing concussion at the joint, and helps to prevent the head of the bone being driven up into the acetabulum.

In an exhaustive article in G. Schwalbe's *Morphologische Arbeiten*, Jena, 1893, Dr. E. Moser reviews the literature of the ligamentum teres briefly and thoroughly. In the bibliography fifty-eight references are given, and the article is illustrated with twenty-seven drawings, principally embryologic.

In the specimens about to be described, the position of the ligamentum teres is well shown in Figs. 5 and 6. These specimens illustrate especially the favorite seats of tuberculous disease, and, taken as they are from a large number of specimens, they may be regarded as fairly representative.

Gray[1] says: "The hip-joint is rarely the seat of acute synovitis from injury, on account of its deep position and its thick covering of soft parts. Acute inflammation may and does frequently occur as the result of constitutional conditions, such as rheumatism, pyemia, etc. . . . Disease of the hip-joint is much more frequently of a chronic character, and is usually of a tuberculous origin. It begins either in the bones or in the synovial membrane, more frequently in the former, and probably in most cases at the growing, highly vascular tissue in the neighborhood of the epiphyseal cartilage. In this respect it differs very materially from tuberculous arthritis of the knee, where the disease usually commences in the synovial membrane.

An examination of Figs. 1, 2, 7, and 8 will show one of the most common seats of tuberculous disease of the hip-joint—namely, the head of the femur. This may or may not be associated with disease of the neck, as is shown in Fig. 1. Or, again, the disease may commence in the neck itself and not affect the head of the bone and the acetabulum, although the two conditions usually go hand in hand. This disease of

[1] Gray's Anatomy, p. 369, ed. 1893, Lea Brothers & Co., Philadelphia.

the neck of the bone alone may go on to such an extent that a complete separation of the head from the shaft results. This is well shown in Figs. 3 and 4.

In excision of the head of the femur for this disease, an operation which is frequently practised at the Children's Hospital, it sometimes happens that the head of the bone with its articular cartilage remains in the acetabulum, and requires the division of the ligamentum teres and some additional dissection before it can be removed.

Fig. 2 shows a case in which complete absorption and disappearance not only of the head but of the neck as well has resulted.

Shortening of the neck of the femur is a frequent result of tuberculous disease. This is well shown in Figs. 3, 6, and 7, as the result of osteitis and necrosis of the bone. It is an almost invariable accompaniment of hip-joint disease, and gives rise at certain stages to the condition of inequality in the length of the two limbs, associated, as it is, with tilting of the pelvis and outward rotation of the head of the bone.

A change of the angle which the shaft makes with the neck of the femur frequently results. This is well shown in Figs. 6 and 7, and is a result of the extension of the tuberculous process either up or down the shaft. Specimens 7 and 8 show how the normal angle of 140°, which the neck makes with the shaft in a healthy femur (see Fig. 5), may be changed to an angle of 128°, or even 110° (Fig. 2). This tuberculous process is one of a rarefying osteitis, and is associated with the formation of pus and the discharge of bone-salts.

In this connection it is interesting to record a typical case of cured coxalgia, which has been under observation for the last nine months. The history of the case is as follows:

Edna K., aged nine years, with a negative family history, except that a maternal aunt died of consumption, had always enjoyed good health until the age of six years, when she had scarlatina and diphtheria at the same time. This was followed by whooping-cough, and the child was so desperately ill that her life was despaired of. In February, 1895, her mother first noticed that she complained of pain in her left knee. It was variously accounted for by different physicians, to whom the child was taken, as growing-pains, rheumatism, and neuralgia. These pains continued, and in April she began to grow lame. She was then restless at night, and frequently cried out with pain in her sleep.

She was seen at the Children's Hospital Dispensary on April 8, 1895, and a diagnosis of incipient coxalgia made. She was advised complete rest in bed, with extension to the diseased leg, and she was accordingly admitted to the Methodist Episcopal Hospital on April 26th. There

FIG. 11.

A case of cured coxalgia with the diseased limb in a position of flexion nine months after treatment was begun.

she remained in bed for four months, with sand-bags on either side of the left leg, and an extension of four pounds in weight. On her admission to the hospital she was in very poor physical condition and extremely pallid. During the four months she remained in bed her color was much improved, and her strength considerably increased. At the end of four months a plaster-of-Paris dressing was applied in place of the extension and rest in bed, and she was allowed to get about with a high shoe on the sound leg and crutches.

Condition of the Hip.—In April, 1895, it was impossible to make the slightest motion of the left hip without causing pain. Four months later there was slight pain on free motion, especially when the knee was flexed on the abdomen. In November, or eight months after treatment was begun, there was very slight fixation on extreme flexion, and no pain whatsoever. This is well shown in Fig. 11, in which the child may be seen standing on her right foot, with the left one elevated in such a way as to bring the knee on the affected side almost up to the abdominal wall. A fixation-splint is to be made for this child, and she will be allowed to go about on crutches until all inflammatory symptoms have disappeared, for six months, when the cautious use of her limb will be allowed. She is now in excellent general condition. She is apparently on the road to perfect recovery, and her case seems to be one of those encouraging ones in which a cure of coxalgia is possible. It is probable that in her case the disease was arrested promptly by the treatment instituted, and, as there was no grave constitutional taint, the routine treatment has been successful. Time alone will decide whether the disease has been successfully arrested. If so, it is probable that the disease was limited to the articular cartilage of the head of the bone, and the inflammatory process arrested before the development of any rarefying osteitis.

We are indebted to Mr. Clifford B. Parker, a second-year student in the Medical Department of the University of Pennsylvania, for the excellent drawings from which figures 1 to 10, inclusive, were taken.

Conclusions.—1. Tuberculous disease of the hip-joint attacks either the head of the femur, the neck of that bone, or the soft tissues in the neighborhood of the articular cartilage, being especially liable to effect the ligamentum teres primarily. It rarely appears first in the acetabulum.

2. Rarefying osteitis of the neck of the femur soon leads to shortening of that portion of the bone.

3. Rarefying osteitis of the neck of the femur allows a change of angle, from pressure, of from 10° to 30°, or even more.

4. The head of the femur may escape disease, and become detached from the shaft if the disease at the neck fail to be arrested.

Discussion.—Dr. G. G. Davis : The subject of tuberculosis of the joints is a very interesting one. I believe that the history will generally show that some injury has preceded the development of the disease. A postmortem injury cannot cause tuberculosis.

Tuberculosis is caused by tuberculosis. An injury may give rise to a simple inflammation, and this injury in an unhealthy person—a person with a tuberculous deposit—may give rise to tuberculosis. In that way I believe tuberculosis oftentimes begins in the joints. There is no doubt that in the knee-joint tuberculosis frequently affects the synovial membrane. The primary cause is in the membrane, and you may find the membrane enlarged and the bones practically intact. In the hip-joint there seems to be less opportunity for membranous disease, but the osseous disease is more marked, though why tuberculosis should begin in the bones primarily is extremely hard to see. One can see why the ligamentum teres, the capsular ligament, and all the soft structures entering into and surrounding the joint should be damaged by an injury to the joint, and why that injury should take on a tuberculous inflammation, yet very often, if not in the majority of cases, tuberculous disease begins in the bone and not in the membranes. It is not at all uncommon to find a tuberculous process oftener in the bone itself than in the superficial part of the joint, where an exfoliation goes on, and this, studded with tubercles, is deposited in the injured part on account of its acting as a *locus minoris resistentiæ*, but why it should be so I cannot see.

From the facts, and the relative importance of the soft parts in diseases of the hip and diseases of the knee, I should be inclined to consider the soft parts more liable in disease of the knee and the bone more in disease of the hip. The bone is more often the primary seat of lesion than the ligament. Dr. Ashhurst's remarks upon the function of the ligamentum teres are very interesting, but I certainly think it is an open question as regards the function of that ligament. It is considered by some to be a retrograde part, and to be like, the vermiform appendix, apparently in process of extinction, and it is perfectly possible that, as in the vermiform appendix, its function may be unknown at present. The resemblance to a coach-spring is certainly a very good one, and, from a mechanical point of view, it does seem to act in that way. When we find children with hip-disease flexing the thigh on the abdomen and abducting it, because abduction and flexion are the two deformities most common in hip-disease ; when we find the victims of hip-disease assum-

ing such an attitude, and that an attitude which tends to put the ligamentum teres on the stretch, it leads me to doubt as to whether it plays the important part which is sometimes attributed to it. It seems to me that, in such cases, the tendency of the child would be rather to relax the ligament and not to put it on the stretch.

Dr. Ashhurst closed by saying: There is one point that I omitted to mention which was recalled to me by Dr. Tunis' reading the extract from Morris's *Anatomy*. Morris maintains his view of the ligament from the fact that he has found it relaxed in almost all cases by trephining the acetabulum. I do not think that we can always assert from our observations on the dead body that a similar condition is present in life. We know that for about 2000 years anatomists maintained that the arteries contained air. I do not wish here to go into a history of this fact. It was not until nearly 2000 years after the arteries had been described that the discovery was made that they contained blood. In those 2000 years the anatomists had only described the arteries from the dead subject. From the time of Harvey the arteries have been described as containing blood instead of air.

In regard to Dr. Davis's opinion, while it is true that tuberculosis manifests itself primarily in the joints, still, at the same time, I believe it is not true that the tuberculosis, or the tubercle-bacillus, really extends to those joints first. I cannot imagine how the bacillus of tuberculosis can make its way into a healthy knee-joint or hip-joint from the external surface, and the only way in which I can account for the occurrence is from the fact, which I think is generally accepted by pathologists, that a very large number of persons—some say almost everyone—have tubercle bacilli somewhere in their body, probably in their tissues, or gone to sleep, as it were, in the glands or in other parts, whence they are roused up. Traumatism usually occurs at the places of least resistance. Although the primary manifestations of tuberculosis are oftentimes in the joints, I do not believe that the bacillus makes its appearance in the joints in the first instance. It must enter through the lungs, or the alimentary canal, or an accidental wound, possibly. It must enter in some way before it can possibly get into the bone or joint. With regard to the effect of traumatism in causing the place of least resistance, and the fact that tuberculous deposits may occur in places not in contact with the soft tissues, I have under my observation at the present time a very interesting case of a man who is the picture of robust health (a surgeon), and who has, at the same time, a very severe and serious tuberculous ulcer of the abdominal wall. While driving around seeing his patients an accident occurred by which he was thrown forward,

striking his abdominal wall. A swelling appeared, which gradually yielded to treatment, and he got well. He had a second accident, and again received a bruise of the same part. After this a lump formed, which slowly softened and finally opened, and it became the seat of a large, tuberculous ulcer. He has had the ulcer scraped and cauterized, and after long and careful treatment his tuberculous ulcer is healing. I believe all tuberculous matter has been removed from it. I have no doubt at all that in this case there was a tuberculous deposit somewhere in his body, though it is barely possible that he may have gotten some on the abdominal wall at the time of the injury. The bacillus might have gotten in there, but it is just as likely he had it before. As the result of this bruise an inflammation followed, and there the bacilli took the opportunity to implant themselves.

In regard to what Dr. Davis said as to the analogy of the appendix, I must confess to be so heterodox as to believe that the vermiform appendix has a function. The late Dr. Harvey taught that it had a function which seems reasonably likely to be the case. Dr. Harvey believed that the function was to secrete a certain amount of mucus, which mucus dropped upon the fecal mass and served the purpose of lubricating it very much as an oil-can would lubricate any machinery to which it was applied. This is a reasonable view, it seems to me. I always like to find a function for organs if I can. In the case of the appendix, we cannot say absolutely that it is entirely without function, and certainly the ligamentum teres I believe has a function, which is explained in the way that I have described. Dr. Davis said very truly that a child with hip-disease may flex the limb upon the abdomen without causing great pain. These cases are those in which the ligamentum teres is not involved. We have pointed out and shown by specimens that in some cases there may be advanced hip-disease without involvement of the ligamentum teres. A child with advanced hip-disease not involving the ligamentum teres no doubt can flex the limb without suffering. That probably is the case in those instances to which Dr. Davis refers. I still believe that the view which I have advanced as to the function of the ligamentum teres is the best one, and is not disproved by the clinical fact which Dr. Davis has told us.

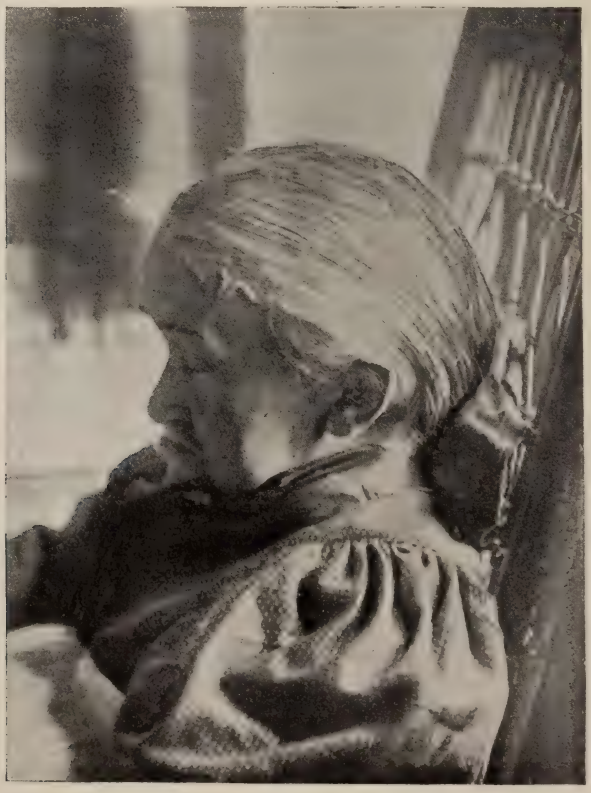
November 14, 1895.

3. *Skull-cap, microscopic bone-sections, and marrow from a case of rarefying osteitis deformans.*

Exhibited by F. Savary Pearce, M.D.

The thickened skull-cap here shown is from a woman, aged seventy-two years, under the care of Dr. Charles Herwirsch, at the "Home for Aged Women." I made the autopsy through the courtesy of Dr. Samuel

Fig. 1.

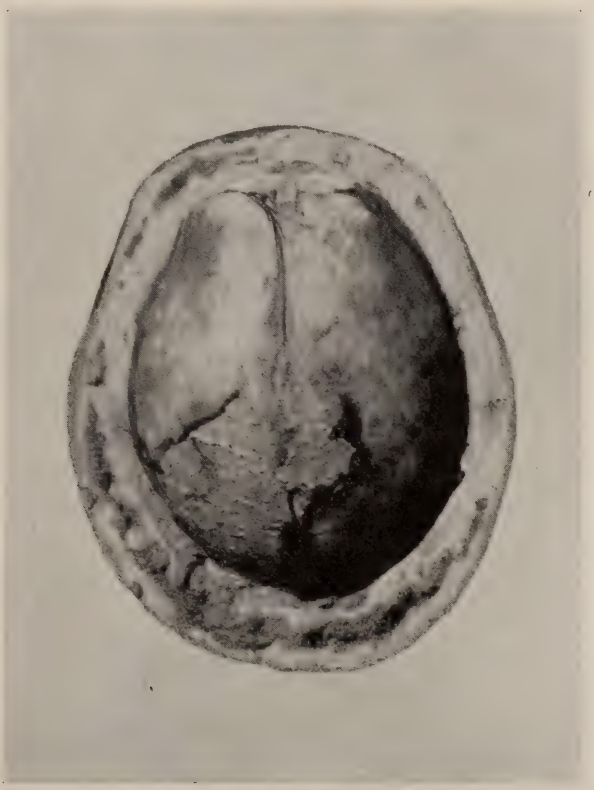


Wolfe, who had been observing the case with Dr. Herwirsch. The autopsy was made twelve hours after death, March 13, 1893. Drs. J. P. C. Griffith, William E. Robertson, and others were present at the section.

The patient could walk up to a short time before demise. There had

never been any paraplegia, contractures, or other resultant of intracranial pressure, although the head was much enlarged, as is well shown in the accompanying photographs.[1] She had been in a condition of asthenia and mental hebetude, which gradually deepened after admission. The woman finally died of the asthenia of old age, influenced by the slow disease-process under consideration. Urinalysis had been negative. No reliable history could be obtained other than that the head-growth had begun seven years ago.

FIG. 2.



The principal thought was that the patient had had a spurious form of hydrocephalus, but the lack of exophthalmos, after the long-continued cephalic enlargement, rather left that possibility *sub judice* until postmortem study.

Postmortem rigidity was well marked. No scars or other signs of

[1] Specimen deposited in the Mütter Museum, College of Physicians of Philadelphia.

abnormal growth presented except in the greatly hypertrophied calvarium and in the slight bowing of the long bones of the lower extremities (most marked in the left leg), as was remarked by Dr. Griffith. The lower half of the head and face was not enlarged. The fingers and toes presented none of the spade-like appearances of acromegaly. The lips were perhaps a little thickened, but there was no true myxedema. Thyroid not palpably enlarged. Skin was everywhere muddy-looking, the characteristic of the prolonged defective nutrition of her body generally.

The temporal arteries were tortuous and somewhat hypertrophied, and veins somewhat distended over the scalp. Hair on scalp very sparse and rather coarse. Occipitofrontal circumference was twenty-eight inches. The bony enlargement of the calvarium was general and symmetric. On making scalp incision there were evidences of venous engorgement throughout. The skin was not thick. The skull-cap was unusually soft and friable, the saw penetrating most readily ; but it was at once noticed that the depth of bone was great and that there was little diploe apparent. The temporal regions were the most dense. On final removal it was found that the vault of skull measured one and three-eighths to one and one-half inches in the occipital region, and three-quarters of an inch in frontal region, with varying differences at the intermediate points. The bone could be easily pricked out with the scalpel. The temporal regions presented an absolute fusing of the once calcareous bone of the internal and external plates. Much lessened diploe in proportion to the greatly increased bone size was apparent in the remaining circumference. In fact, little diploe was seen, excepting in the occipital bone, the thickest part of the skull. The basilar process of the occipital, the body of the sphenoid, and other bones of the base of the skull, were very easily penetrated by the chisel. There was nothing pathologic in the brain membranes, further than that the dura was slightly thickened. The brain itself weighed forty-eight ounces, was not enlarged, fitting nicely into the normal-sized cavity. The ventricles were distended beyond the normal by clear serum. Ependyma normal. No hemorrhage, growth, or other alteration was found in the encephalon. Thus the head enlargement was entirely bony. What was the nature of the process? The necessity for further search in other bones became clearer.

Both femora were found thickened and slightly curved with convexities forward and outward. The epiphyses were much enlarged and covered with a new growth, a capping, as it were, of osteoplastic incrustation (exostoses) of friable, mealy bone.

The left tibia was greatly thickened, and curved forward and outward. The medullary cavity was disproportionately enlarged, and was filled completely with red marrow, some of which, together with the adjacent bone, was removed for microscopic study. The right tibia was little affected. The bones of the arms, hands, and feet were not noticeably enlarged, excepting a slight protrusion and thickening of the calcanea posteriorly.

Median abdominal incision revealed much yellow fat beneath the skin and in the omentum.

The ribs proved also to be soft and friable, and cut readily with the ordinary scalpel used in the section. Here, also, the bone-marrow had fetal characteristics. The crests of ilia were more blunted and larger than normal. Pubis the same. Portions of these were chiselled off for study. The pleural and pericardial sacs contained normal amounts of fluid and were free throughout.

Aside from slight emphysematous areas anteriorly and above, and postmortem congestion at the bases behind, the lungs presented no pathologic changes. The heart was fatty and slightly hypertrophied. There was a very large and dense chicken-fat clot extending into the aorta from the left ventricle; also the same kind of clot adhering to the mitral leaflets and their chordae tendineæ, demonstrating the slow form of death. Atheroma existed at the base of the posterior mitral leaflet. The mitral orifice admitted two fingers readily. In the right heart there was also found much whipped clot and less currant-jelly clot. The tricuspid opening readily admitted three fingers. The wall of the right ventricle contained more fat than muscle-fibers. The liver was normal in size, and was the seat of a slight degree of cirrhosis and fatty degeneration. The kidneys were slightly enlarged, cortices somewhat narrowed. The capsule stripped readily; beginning interstitial change was evident. The spleen was slightly reduced in size. The intestines and other abdominal viscera were normal.

Just where this case belongs in pathology is not so easy to decide. The name "Rarefying Osteitis Deformans" suits for most of the bony changes, for there is certainly porosis in places, with deformity.

Slides of bone from the pubis and femur, made by Dr. Robertson, show absence of bone-cells, the evidences of chronic inflammation and irregularly enlarged Haversian systems. The sections of the bone and marrow, kindly made from the left tibia for me by Dr. A. E. Taylor, after decalcifying, hardening and staining by fuchsin and thionin, show a queer lot of changes, viz., atrophy of bone, a great loss in the number of osteophytes and osteoclasts, and conversion of parts of

the bone into mere fibrous structure. The marrow shows very few myelocytes.

It would seem that the changes here found indicate an advanced state of a widespread skeletal disease in which detritus was the remnant. The bone was decalcified (with 2 per cent. HCl in 70 per cent. alcohol) in twenty-four hours, not in a week, as is the case with normal bone.

What, then, is this widespread bone affection? Rickets is, of course, out of court, as this disease, while having curved bones, never has such enormous enlargement, and it occurs in youth. In mollities ossium the bones are decalcified, elastic, and flexible; therefore not friable, sclerosed, or brittle. In acromegaly the lower part of the head is mainly enlarged (not the calvarium). The jaws are somewhat prominent. The hands would present the bony growth. In any event the head would never attain the extreme hypertrophy shown here; and the pituitary body would likely have been affected in acromegaly. In leontiasis ossium, so-called, one would expect firmer sclerosis than was present in this skull-cap, which was the most dense (and yet less dense than normal bone with all its thickness). The malar prominences would have been prominent in leontiasis cranii, and, as inferred, eburnation predominates, not friability. May not leontiasis, after all, be the further condensing osteitis in which the malar bones also become affected?

This extreme bony enlargement is a very rare occurrence, and its association with the widespread bone disease I cannot find recorded anywhere. The weight of evidence seems to be in favor of a chronic, rarefying osteitis, with deformities, hypertrophy, and a final condensation, as is well shown in the skull. The low ebb of myelogenesis, as pointed out by the absence of myelocytes in the bone-marrow, would have made the blood study during life of interest.

Dr. M. Allen Starr, in the *American Journal of the Medical Sciences*, for December, 1894, reports a case of hypertrophy of the skull, also with some enlargement of the vertebræ. Virchow gives an analysis of some of the bony enlargements in his treatise on tumors, and considers the process inflammatory.

A monograph by Baumgarten on *Leontiasis Ossea*, Paris, 1892, completes the principal literature of this affection of the skeleton.

Dr. James J. Putnam reported four cases of what he termed "Hyperostosis Cranii" (cephalomegaly) to the American Neurological Society in June, 1895. The rest of the bones of the body were not involved. These cases of head-involvement alone Dr. Putnam divides into the (*a*) upper-head type; and (*b*) the lower-head type, where the malar bones become involved and produce the leonic appearance. Two of the cases

were in females. In one woman, aged thirty-five years, the enlargement had begun at the twelfth year; in the other, at the nineteenth year. In one of the males, aged fifty-three years, the growth had begun at about the fifteenth year; in the other it began in infancy. The four are spoken of as dystrophies.

Discussion.—Dr. Griffith said that Dr. Pearce had asked him to see a case of hydrocephalus in an old woman. At the autopsy he saw that Dr. Pearce had considerable difficulty in sawing the skull. On examining the skull he noted that the malar bones were very large and the upper part of the face was much larger than the lower. There was a decided disproportion between the two sides of the face. The growth within this bone was hard in its nature and very heavy. He had read of conditions similar to this where at first soft bone was present and afterward hard bone. Perhaps the growth of new bone occurred from the periosteum. All of the changes in the skull seemed to run into each other, and one could not ascertain the relations that the parts bore to each other. The whole skull probably was at one time in a condition of sclerosis. He supposed the case might be called one of osteitis deformans. The condition was very rare. Paget had described five cases of it, but very few cases were reported, and the specimen was a very interesting one. *April* 9, 1896.

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4. *Specimens from a case of arthritis deformans.*

Exhibited by J. M. Swan, M.D.

The subject from which these specimens were taken was a male, aged about forty years. On the dissecting table at the University of Pennsylvania multiple osteomata were noticed along the ventral surface of the bodies of the vertebra. I imagined that the articular ends of the femora which entered into the formation of the knee-joints were increased in size. This led to a careful examination of all the bones of the skeleton. Unfortunately, no history of the case before its appearance in the dissecting-room can be obtained.

Contrary to expectation, it was found that the lower extremities of the femora were not disproportionately enlarged. On disarticulating at the hip, the upper extremities of both femora were found to have undergone marked changes. The neck of the left femur (Figs. 1 and 2)

FIG. 1.

Left femur, posterior surface.

FIG. 2. FIG. 3.

Left femur, anterior surface. Right femur, posterior surface.

has been reduced by a process of softening and absorption until the distance from the shaft of the bone to the head is only 1.5 centimeters. The summit of the great trochanter projects sixteen millimeters above the highest point on the head of the bone. All the facets for the attachment of muscles present lips of bony material which form distinct cavities. These hyperostoses are also noted along the upper portion of the linea aspera. The neck looks as though it had been forcibly twisted when hot, and allowed to cool in the new position, and about the head of the bone are those overhanging masses of osseous substance which appear as though melted material had been poured over the bone and then allowed to cool. These formations are called by Warren[1] "molten" fringes. The fossa for the insertion of the ligamentum teres is so dislocated that it occupies a position on the lower and anterior portion of the head. This fossa is larger than normal and its margins are much roughened. The entire head of the bone is flattened and broadened, and the articular surface is eburnated.

The right femur (Fig. 3) presents the same characteristics as the left, but in much less degree. The upper limit of the great trochanter projects eleven millimeters above the upper point of the head. The facets for insertion of the muscles present the same bony lips as those noted on the other bone, and these hyperostoses also affect the upper portion of the linea aspera. The neck of the bone appears twisted and forms a smaller angle than normal with the neck. The molten fringes about the head are present, and the fossa for the ligamentum teres is dislocated. Both bones seem increased in size, and the lower articular extremities, while not presenting any marked signs of disease, appear to be clumsy. Both innominate bones (Figs. 4 and 5) are large and present numerous exostoses corresponding to the positions of the origin and insertion of the various muscles. These formations are most numerous along the crests of the ilia, along the rami of the pubes and ischia, and on the tuberosities and the spines of the ischia. The acetabula, of course, present the most marked deformities, corresponding to the diseased heads of the femora. The left bone (*a*) presents a cavity which is seven centimeters wide by eight centimeters long. The rough area at the bottom of the cavity for the attachment of the ligamentum teres is entirely wanting, and its place is taken by a mass of osseous tissue, which looks as though it had been run in while it was hot and then allowed to solidify. The surface of this mass presents several facets which are separated by sharper or blunter ridges, and which seem to be formed by

[1] Warren: Surgical Pathology and Therapeutics, Philadelphia, 1895.

the different degrees of pressure exerted at various points. This bone is eburnated and highly polished. The cotyloid notch is narrower than normal, and the edges of the entire cavity are thickened and raised by the molten fringes. The right acetabulum (*b*) measures 6.5 by 7.5 centimeters. The articular surface is increased in extent, and the cavity is more shallow than normal. The rough surface for the attachment of the ligamentum teres is not entirely obliterated and presents numerous ridges of new bone-formation. The surfaces for articulation with the sacrum are larger than normal and present several nodular excrescences.

FIG. 4. FIG. 5.

a *b*

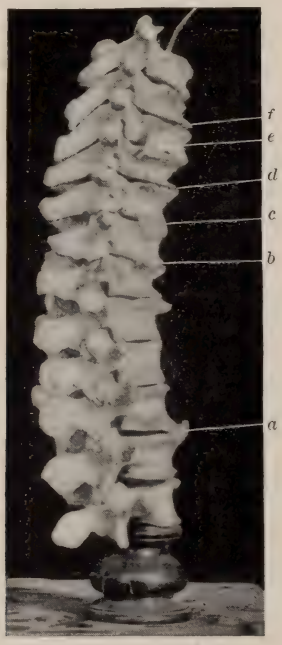
Innominate bones.

The articular surfaces forming the symphysis pubis are increased in size, being broadened. The sacrum appears light and spongy. Exostoses are not numerous. The left articular surface is broader than the right.

The changes in the vertebral column (Fig. 6) are most marked in the lower thoracic region, although all the bones are affected to a greater or less degree. There is a slight lateral curvature with the convexity to the left in the lumbar and lower thoracic regions, and a compensatory curvature with the convexity to the right in the upper thoracic and cervical regions. There is also an anteroposterior curvature due to the diminution in the size of the bodies of the eighth, ninth, and twelfth

thoracic segments. The bones seem to be rarefied. Exostoses are numerous and in some places are very marked. The molten fringes are seen between the adjacent portions of the bodies of the bones, ventrally and laterally. The outgrowths of bony substance seem to take place equally from the cephalad and caudad bone, and the two lips meet over the intervertebral disk, forming, in the recent state, a long tumor of pyramidal shape. These formations are most marked between the third and fourth lumbar segments (Fig. 6, *a*), and between the eleventh and

FIG. 6.



Vertebral column seen from right side.

twelfth (*b*), tenth and eleventh (*c*), ninth and tenth (*d*), eighth and ninth (*e*), and seventh and eighth (*f*) thoracic segments. Nearly all the articular surfaces are deepened by outgrowths of bone. These hyperostoses above the sixth thoracic segment are less numerous and less robust. The articular facets for the ribs on the bodies and transverse processes of the thoracic segments are distorted and misshapen. In the lumbar region, particularly, the tips of the spinous processes are deformed by bony outgrowths.

The heads and necks of the ribs are distorted and their articular facets variously ornamented with exostotic projections.

On first examination of these specimens four possibilities suggest themselves: the case is one of acromegaly, of syphilis, of osteitis deformans, or of arthritis deformans.

The absence of involvement of the bones of the hands and feet and of the face, together with the existence of a pituitary fossa of normal size, would seem to exclude acromegaly. The appearance of the subject would also serve to exclude this condition.

Syphilitic lesions of the bones are characterized by localized swellings or nodes projecting from the surface of the diseased bone and by areas of caries or necrosis. The formation of these lesions may follow acquired or inherited syphilis, irrespective of the growth of a gumma (Macnamara[1]). The absence of carious or necrotic lesions and the freedom of the shafts of the long bones from involvement in the process would argue against the specific origin of the disease.

Osteitis deformans, according to Ziegler,[2] is a disease of old age: this subject was not much beyond the forty-year mark. The bones of the skull are usually involved in the disease; the skull in this subject was quite normal in appearance, the bones being of proper thickness and presenting no exostoses. In osteitis deformans, also, the process involves the shafts of the bones, producing bendings, angular flexures, and various other striking deformities. In these specimens it will be seen that the shafts of the long bones are quite straight and that they present no evidences of disease, except for the bony formations at the muscular attachments. The vertebral column, it is true, presents a picture which is indicative of this disease; but, it will be seen later, this appearance is also characteristic of arthritis deformans. On account of the appearance of the long bones I have excluded osteitis deformans.

But one condition now remains, and I have diagnosed these as specimens of that condition, arthritis deformans, partly by exclusion and partly because the lesions are almost exclusively in the joint regions. The appearance of the left femur and of the left acetabulum indicates that this process is one which has involved the entire structure of the joint and in which the balance between bone-absorption and bone-deposition has been markedly upset.

It is quite unfortunate that the bones were beyond microscopic possibilities when first seen. But, in order to give an idea of the processes

[1] Macnamara: Diseases of Bones and Joints, third edition, London, 1887.

[2] Ziegler: A Text-book of Special Pathological Anatomy, translated and edited by Donald MacAlister, M.D., and Henry W. Cattell, M.D., New York, 1896.

which have been at work to produce these remarkable architectural features, I will quote Ziegler.[1] The changes in the cartilages consist of fibrillation and cleavage or splitting of the superficial layers; to this is usually superadded extensive softening, with the consequent formation of cavities in the deeper layers next the bone. Along with these degenerative processes free hyperplastic proliferation takes place, and often leads to nodose thickening of the cartilage. The deeply-situated cavities due to softening are sooner or later lined by vascular medullary tissue which grows up from the bone. The substance of the cartilage itself is often directly permeated by the growing marrow. When the deeper layers of the cartilage are thus traversed in all directions by medullary spaces, the remaining islands and bridges between these are generally converted into osteoid tissue and ultimately into calcified bone. The changes in the bones are for the most part retrogressive, and are chiefly of the nature of resorption; they lead to lacunar atrophy of the osseous trabeculæ, and sometimes entire lamellæ are destroyed, so that the bone gives way at the affected part. In the course of years these changes lead to very marked deformities of the joints. Proliferation of the cartilage with subsequent ossification occurs mainly at the periphery of the articular head and of the articular cavity. In the former situation it gives rise to tuberous excrescences, and in the latter it causes the socket to be wholly or partially encircled with a ridge by which the cavity is often notably enlarged and deepened. The central parts of the articular head that are most subjected to pressure and friction are usually flattened, while the articular socket becomes wider. All of these changes take place whether the articular cartilage is preserved or not, and in the former case depend upon a subchondral atrophy of the bone, as a result of which the cartilage is undermined and collapses. If the cartilage becomes fibrillated and destroyed, the underlying bone is, of course, exposed, and, in particular, those parts of it that have been newly formed by subchondral ossification. Such bone is often very dense, and in places has the compact texture and appearance of ivory. If the affected limb continues to be movable, the hard bony surface of the articular head, as a result of constant movement, is often polished smooth; or if the movements take place in one plane only, it is marked with parallel grooves.

In these specimens all of these changes have evidently taken place, and the condition is an old one. The resorption-processes in the bone have caused the neck to become displaced and contorted; the growing

[1] Ibid.

of the marrow into the softened cartilage has formed the molten fringes which line the margins of the articular cavities, which border the articular heads of the bones, and which give the vertebræ the appearance known as spondylitis deformans. Later, the cartilage in the hip-joint has become destroyed, the articular head of the femur and the acetabulum have moved, the one on the other, which has resulted in the polishing of the surfaces, the formation of ridges, flattening of the parts, and eburnation.

Absence of involvement of the bones of the cranium, the symmetric development of the lesions, the absence of general osteosclerosis, and the marked deformity are further reasons for considering the processes which have produced these lesions as those of chronic arthritis deformans. In this disease, too, we find the most remarkable varieties of hyperplastic overgrowth and the most marked degenerative changes in the cartilages and bones.

How account for the exostoses along the muscular attachments? It is in every way probable that the resorption of bone in various portions of the skeleton has left the blood so overcharged with lime-salts that the disturbance caused by the constant action of the muscles has been sufficient to produce these growths from the deposition of the circulating bone-material.

Discussion.—Dr. A. A. Eshner asked whether Dr. Swan had observed the condition of the hands and feet in this case. The lesions of arthritis deformans were rather peculiar in these parts in that deflection always took place toward the ulnar aspect.

Dr. Swan replied that the hands and feet were examined, but were not involved. As he had only seen the case at autopsy and not clinically, he could not say whether or not there was any stiffness during life.

Dr. F. A. Packard thought the case very interesting, especially since reading Adams' book. Balantine had recently found a micro-organism in this disease, which he claimed was the cause of the affection.

Dr. J. McFarland remarked that in Orth's *Pathologische Diagnostik* there was a picture quite like the case of Dr. Swan's.

Dr. Swan, in closing, said that there was also a picture in Ziegler's book quite similar to this, except that his case was more marked, especially as regards the femur, than the one figured by Ziegler.

October 22, 1896.

5. *Superficial caries of the femora, tibiæ, and fibulæ.*

Exhibited by J. M. Swan, M.D.

The bones present extensive and irregular areas of superficial caries. The process is most marked in the right limb. On this side of the body the posterior surface of the tibia is the farthest advanced in the disease. The entire bone is thickened, and the irregularity of its surface indicates the process of lacunar atrophy which has taken place. At the articular ends of the bones of the right side, particularly at the lower end of the femur, and at the upper end of the tibia, bone-apposition is indicated by tuberosities of new bone-tissue of varying sizes. The spine of the tibia is converted by this process into a wide and thick rounded eminence, which must have interfered with the movement of the joint. The etiology of this condition can only be stated in the range of possibilities, as nothing is known of the history of the case. Causes of superficial caries are syphilis, tuberculosis, and the acute infectious diseases. The probabilities are that these bones are tuberculous. *March* 11, 1897.

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6. *Specimens of tibiæ and fibulæ showing the deformities produced by rickets.*

Exhibited by J. M. Swan, M.D.

The bones were taken from the body of a negro adult, aged about twenty-five years, which found its way to the dissecting-table at the University of Pennsylvania.

The right tibia is much more markedly diseased than the left. Both bones show marked anterior curvature and external twisting. The fibulæ are comparatively straight, the shafts being only slightly twisted. The disease in these bones has evidently been of a mild character, and confined to the diaphyses of the bones, and was never of so severe a character as to keep the patient in bed. The softened bone due to lacunar absorption could not well bear the weight of the individual, and the prominent curves resulted. Subsequently the progress of the disease was arrested and the firm osseous tissue was deposited on deformed lines previously mapped out. *March* 11, 1897.

II. THE DIGESTIVE TRACT.

1. *Tuberculous ulcer of stomach.*

Exhibited by F. A. Packard, M.D.

The specimen presented was found at the autopsy performed upon a little mulatto girl, aged four years, who was admitted to the Children's Hospital on August 16, 1895, and died on October 23d. During life she presented the typical appearance of combined rachitis and tuberculosis, had marked signs of pulmonary tuberculosis, but had no gastric symptoms save anorexia. The latter was not more than would be accounted for by her primary disease. She gradually failed in spite of being sent to the country-branch, and died of exhaustion.

At autopsy, on opening the abdomen, quite a large quantity of grayish-yellow, purulent fluid was found, amounting in all to about six to eight ounces. The diaphragm on the right side was at the level of the fourth rib, on the left at the level of the fourth interspace. The great omentum was rolled up, and presented on its anterior surface two large pearly nodules, measuring 3 mm. in diameter. The gastrocolic omentum was filled with mucoid material of a yellowish-white color. The mesenteric glands were plainly visible as large, white nodules projecting between the coils of small intestine. On the anterior surface of the caput coli numerous tuberculous nodules were to be felt and seen. The liver projected 4.5 cm. below the costal margin in the right nipple line, 5 cm. below the ensiform cartilage in the middle line.

In the anterior mediastinum were numerous hard, irregularly-shaped masses adherent to the posterior surface of the sternum. In the right pleural cavity there was a considerable amount of grayish, turbid fluid. At the upper portion of the right lung there were moderately firm adhesions, especially posteriorly. On the left side there was a small amount of clear, serous fluid, and a few adhesions were present at the apex.

Between the pericardial sac and the diaphragm there was quite a bunch of enlarged, hard lymph-nodes.

The pericardium contained about an ounce of clear serum. The parietal layer of the pericardium appeared to be normal. The right

auricle, left auricle, and left ventricle contained chicken-fat clots, but the cardiac examination was otherwise negative. The glands of the posterior mediastinum were greatly enlarged, and chains of enlarged lymph-nodes could be traced upward on both sides of the trachea and around the aorta. Both lungs showed marbling from the presence of atelectatic areas. On the pleural surface of both lungs there were many small, pearly nodules, giving the surface a roughness to the touch. Both lungs, on section, showed a great number of small, pearly-white nodules of about the size of a pin's-head scattered throughout their structure. In addition, in the right lung there was found a large, yellowish-white mass, irregular in shape, measuring 1 cm. by 2 cm. in diameter, of cheesy consistence. On the right side the parietal pleura showed two small masses of yellowish-white color, one being situated on the costal, the other on the diaphragmatic surface. The peritoneum, as a whole, was fairly smooth, but on the parietal layer many tuberculous nodules were found.

The spleen measured 9 by 4.5 by 1.5 cm. Its capsule was much thickened. At its upper edge one small tubercle was found, but the remaining thickening appeared to be due to nontuberculous perisplenitis. On section, a few white nodules were seen disseminated through the parenchyma.

The suprarenal capsules were normal, as were also the kidneys. About the notch of the liver there were many enlarged lymph-nodes, which did not appear to obstruct the vessels or ducts at the notch. The liver and pancreas were normal.

The stomach was of average size. Its mucous membrane looked rather thickened, and at the time of autopsy was covered with glairy mucus. On the posterior surface near the cardiac orifice there was seen a crater-like depression with raised and indurated walls. The center of this area of disease was covered with pale, granulation-like material, looking like a simple ulcer of the stomach of the adult. The whole area involved measured 1 by 1.5 cm. The borders were perfectly smooth.

In the caput coli there was a large sloughing ulcer with gangrenous-looking walls. The shape of the ulcer was irregular, and it was directed spirally around the wall of the gut so as to involve the whole width. In the ascending colon, about 10 cm. above the cecum, there was the scar of an old, healed ulcer. In the small intestine there was found but one ulcer, which was situated in the upper part of the ileum.

On the peritoneal surface of the intestines and stomach there were small pin-head tubercles corresponding to the ulcers and to the scar-tissue mentioned as present in the ascending colon. Behind the stomach,

corresponding to the position of the gastric ulcer, there was a small, enlarged lymphatic gland, firm on section, and distinctly separated from the serous coat of the organ.

Saddling the lumbar vertebral bodies there was a bunch of greatly enlarged and welded lymphatic glands.

Section of the gastric ulcer shows the presence of giant-cells, small-cell infiltration, and tubercle-bacilli.

The rarity of ulcer of the stomach, other than the so-called hemorrhagic erosions, in childhood, and the rarity of tuberculous gastric ulcer in subjects of all ages, make this specimen one of rather more than average interest. *October* 24, 1895.

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2. *Perforating typhoid ulcer of appendix.*

Exhibited by W. S. Carter, M.D.

The specimen was obtained at the autopsy on a male child, aged eleven years, that had been admitted to the Children's Hospital on September 9, 1895. The boy had been ill about a week before his entrance into the hospital, with abdominal pain, loose bowels, and fever. The abdomen was slightly tympanitic, but there were no spots or splenic enlargement. The temperature varied from 103° to 105° F.; on the 13th it began to fall, the pulse remaining weak; delirium continued, and retention of urine set in. On September 18th the temperature sank to 97.4° F., and the pulse became rapid and feeble. The boy died in seven hours, without developing any abdominal symptoms. The temperature rose to 102° F. before death.

At the autopsy a perforated typhoid ulcer was found in the appendix. *October* 24, 1895.

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3. *A case of congenital absence of the vermiform appendix.*

Exhibited by J. M. Swan, M.D.

The specimen which I present was taken from a male subject in the dissecting-room of the University of Pennsylvania. The apparent age of the individual was forty years.

On examining the external aspect of the cecum, as it lay in the body, no trace of a vermiform appendix could be found. The intestines were

then removed, and a careful search instituted, but still no tissue was discovered which in any way resembled the appendix. The longitudinal bands of muscular fibers were traced down to their termination, and the wall of the bowel presented a perfectly smooth appearance, the serous covering of the gut presenting no cicatricial tissue, thus indicating that this portion of the intestinal canal had not suffered at the hands of the surgeon. On examining the cecum from its internal surface the ileocecal valve was seen to be normal in situation and appearance. The normal situation of the vermiform appendix is on the posterior wall of this portion of the intestine, just internal to the position of the ileocecal valve, and on the mucous membrane of the bowel is seen a dimple marking the opening into the lumen of the appendix. No such dimple could be detected in this specimen. Situated below the orifice of the ileum is an oval ulcer, which has involved the coats of the bowel down as far as the serosa. The base of this ulcer is smooth and shining, the margins are elevated, and for some distance from the raised border there is presented an appearance resembling that of several miliary tubercles. A second ulcer, similar in appearance, is seen at the apex of the cecum. Owing to advanced postmortem changes, microscopic sections, which were made, failed to show the characteristic appearance of a tuberculous ulcer, and the diagnosis of tuberculous ulcer has to be made from the macroscopic appearance alone. The abdominal wall presented no scar which would point to the performance of a celiotomy at any time.

Taking these facts into consideration, I call this a case of congenital absence of the vermiform appendix. The explanation of the condition is that the head of the colon has developed equally in all directions, thus including in the cecum that portion of the intestine which is usually undeveloped, and which represents the lumen of the large intestine as it is first formed.

In reviewing the literature of the subject, I find a case reported in which the appendix was absent in a male child that died, four days after birth, from intestinal obstruction. In this case, however, at autopsy, the small intestine ended in a blind pouch at the lower end of the ileum, and there was no connection between it and the large intestine at any point corresponding to an ileocecal valve.[1]

Struthers, in his paper "On the Varieties of the Appendix Vermiformis, Cecum, and Ileocecal Valve in Man,"[2] mentions no case of con-

[1] J. C. Oliver, M.D., in Cincinnati Lancet-Clinic, vol. xxvii. p 203.
[2] Edinburgh Medical Journal, vol. xxxix. Part I, p. 289.

genital absence of this portion of the alimentary canal. This paper gives, as the shortest specimen seen, a small rudimentary process three-quarters of an inch in length. Struthers also mentions a case in which the orifice of the appendix was entirely closed, and in which, on the back of the cecum, was a small thickening, like a stump, indicating the position from which the appendix had arisen. This condition was evidently due to retrograde changes in the organ.

Piersol[1] gives as the extremes of length of the appendix seventeen millimeters (Zuckerkandl) and 230 millimeters (Luschka); but he mentions no case in which there was an entire absence of the process.

Wharton[2] reports cases of congenital absence of cecum and a portion of the colon, but no case of the presence of a well-developed cecum with absence of the appendix vermiformis.

Dr. Guitéras[3] has seen one case occurring on the autopsy-table at the Philadelphia Hospital, in which the vermiform appendix was congenitally absent. This case, however, has not been reported.

Kelynack[4] says that the vermiform appendix is a constant structure in man, but not in the higher animals generally. In speaking of the variations in size, Kelynack[5] states that "complete absence of the appendix has been recorded by Ferguson and others." In referring to Ferguson's paper,[6] I find that the author made 200 careful dissections of this portion of the body, the cecum, and "in one instance only could it (the appendix) be said to be absent."

Kelynack[7] quotes Darwin[8] as saying, "it (the vermiform appendix) is occasionally quite absent." He further quotes Treves[9] as saying, "it is quite common to find, in postmortem subjects, that the vermiform process is wanting." This statement of Mr. Treves, however, seems to be too sweeping, and not founded upon the facts of the case. Taking into consideration the few instances reported in literature, it would seem that the condition is far from common. The conservative words of Darwin seem to be better applicable to the case, and we may still, even in light that closer attention to this region has shed upon our knowledge, say that the vermiform appendix is "occasionally" not to be found.

[1] "Obliteration of the Appendix Vermiformis." University Medical Magazine, vol. vii. No. 12, p. 893.
[2] Keating's Cyclopedia of the Diseases of Children, vol. iii. p. 315.
[3] Personal communication.
[4] A Contribution to the Pathology of the Vermiform Appendix, London, 1893.
[5] Loc cit.
[6] American Journal of the Medical Sciences, January, 1891.
[7] Loc. cit.
[8] The Descent of Man, second edition, 1874, p. 21.
[9] The Surgical Treatment of Typhlitis, 1890, p. 140.

I am indebted to Dr. Frank D. Kilgore, who was dissecting the subject, for calling my attention to the specimen. *October* 24, 1895.

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4. *Carcinoma of the colon.*

Exhibited by T. S. Kirkbride, Jr., M.D.

The specimen of carcinoma of the colon which I present to-night, is one which was removed by Dr. T. S. K. Morton at the Pennsylvania Hospital from a patient referred to him for operation by Dr. Musser and Dr. Lowree, of Tyrone. As a report of the case will be made by Dr. Musser and Dr. Morton, I shall merely outline the patient's history and the nature of the operation before describing the specimen itself, which, by the way, is made doubly interesting, owing to the fact that the patient from whom it was excised is at present enjoying good health.

The patient, a grain-broker, aged fifty-two years, had measles and whooping-cough when a child. In 1885 he had a slight attack of inflammatory rheumatism, and a severe attack during the winter of 1889–90. Otherwise, except for so-called nervous attacks, to which he has been subject for twenty years, he has been well and active until the present illness. The above attacks, which he now attributes to the bowel trouble, consisted of nervous feeling in the back, with vertigo and cold extremities.

The first attack of pain in the left side of abdomen occurred about twenty years ago. The pain was dull and aching in character, and was relieved by counterirritation, but at times during the following years recurred with greater or less severity. During the last two years the pain has been more constant and more severe, but never enough to give any great discomfort.

For nearly fifteen years his bowel movements have been very small in diameter, and during the past eight months never larger than 1 or 1½ cm. There has been a constant feeling of fulness of the bowel on the right side, with a sense of obstruction on the left. About five years ago he first noticed blood in the movements; it was then present for about three weeks. Again, eight months ago, he passed clotted blood in considerable amount, the blood being passed especially while the bowels were inactive, and ceasing when the latter were freely moved by purgatives, which, for several years, have been required to produce a movement. During the past eight months about twenty pounds have been lost in weight, which at present is about 145 pounds.

Physical examination. The patient is fairly well formed, but rather emaciated. The complexion is pale and the general appearance cachectic. Inspection of the abdomen was negative. On palpation there was felt in the left hypochondrium an indistinct mass just projecting below the margin of the ribs. The mass is most distinct after the bowels have been freely moved by purgatives. Percussion-resonance is impaired in the left hypochondrium.

Rectal examination was negative. There was no enlargement of the inguinal glands.

The tumor was removed through an incision in the left linea semilunaris. It was found to be a neoplasm of the gut (colon), situated just below the splenic flexure, and affecting the entire circumference, but apparently entirely localized and without any infiltration of the mesenteric glands. The entire mass was delivered through the abdominal incision. The mesenteric attachment was divided for about 14 cm. and a portion of the gut about 18 cm. long excised. This included 5 cm. of healthy bowel above and below the infiltrated portion. The anastomoses was effected by means of a Murphy button—end-to-end anastomosis.

The specimen, which is unfortunately somewhat distorted by hardening in alcohol, showed, when fresh, a tumor of 8½ cm. long, between 1 and 2 cm. thick, and infiltrating the entire circumference of the bowel. The lumen of the gut was entirely closed, except for an irregular opening about 1 cm. in diameter.

On dividing the mass its surface was found to be rough and irregular, with patches of ulceration and superficial necrosis. The gut above the obstruction was considerably dilated and the wall thin; below, the lumen is somewhat contracted. The consistency of the tumor is moderately firm. Its general appearance is that of a large-celled carcinoma.

Microscopic section from the lower junction of tumor and healthy intestine shows quite well the gradual transition from the normal structure of the glandular epithelium to its most atypical formation in the substance of the tumor, which is a beautiful example of a cylindric-celled carcinoma.

The internal surface shows an area of necrosis backed by a line of cells with fragmenting nuclei, and these in places followed by true granulation-tissue with an attempt at the formation of new bloodvessels. Throughout the tumor there are many polynuclear leukocytes scattered among the epithelial cells.

The connective-tissue stroma is quite scanty.

In connection with this specimen there is another one from a typhoid ulcer of the intestine, which I show as being interesting in connection

with the discussion of the etiology of new epithelial growths in the intestines. The section to which I refer shows at the margin of a healing ulcer an epithelial growth which has a striking resemblance to the edges of the carcinoma of the first specimen. *November* 14, 1895.

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5. *Cirrhosis of liver.*

Exhibited by H. M. Fisher, M.D.

This specimen is a liver that was removed last Saturday from the body of a patient who had presented the symptoms of cirrhosis of the liver. He had suffered from lead-poisoning some three or four years ago, and was admitted to the hospital about a year ago, shortly afterward leaving slightly improved. He came under my care last summer, when he showed symptoms of cirrhosis with mild ascites. He complained of much pain over the region of the liver. The organ shows marked induration and great thickening of the capsule. The whitish-yellow indurations were much better seen in the fresh condition. I think it has been the subject of sarcomatous infiltration. No sections have as yet been made. There is no evidence of sarcomatous infiltration of any of the other organs. *November* 14, 1895.

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6. *Cystic gall-bladder—tuberculosis—nephritis.*

Exhibited by M. H. Fussell, M.D.

The patient from whom these specimens were removed was an inmate of Hayes Mechanics' Home, in the care of Dr. Charles Service, of Bala, to whom I acknowledge my indebtedness.

The history was extremely meagre. Nothing could be obtained save that during the man's residence at the institution he had constantly suffered from severe gastric disturbance, with jaundice, during the last few weeks of life.

I saw him when moribund. He was much emaciated, jaundiced, and complained constantly of vomiting and nausea, accompanied with severe pain in the epigastrium.

On examination, the abdominal walls were much contracted. Lying in the right inguinal region was a pear-shaped mass. The mass was

smooth, apparently cystic, and extremely movable. It could be placed in any position in the abdomen—at pubis, to left or above, without giving any especial distress to the patient. From the body of the mass a long pedicle could be traced leading up to the liver.

A diagnosis of a cystic gall-bladder was made, with the probable cause of the occlusion, based on the meagre history of a carcinomatous occlusion of the neck. The liver was not enlarged. No other mass could be detected.

At the postmortem, forty-eight hours after death, the following conditions were found:

Right lung crepitant throughout, except at the extreme apex, where there was a consolidated area, which was largely central, being surrounded by emphysematous lung-tissue. Left lung emphysematous, and in the region of the root a consolidated area having scattered through it cheesy areas. Heart dilated; shows postmortem softening. No valvular lesion. Spleen small; capsule thickened; no miliary bodies found either in or upon it. Right kidney atrophied and tightly adherent to perinephric tissue. It measures 3 by 1½ inches, and weighs two ounces. The pelvis is large in comparison with the kidney-substance. There is scarcely any cortical substance remaining. The capsule is adherent, and on being separated tears off portions of the kidney-substance. Left kidney enlarged and weighs eight ounces. Its capsule is adherent. There are two small cysts upon its surface filled with clear liquid; a few miliary tubercles are also scattered over its surface. A supernumerary artery enters its upper end, and a second its lower end. The center of the kidney is filled with a large quantity of fat. The cortical substance is much atrophied.

The liver is rather smaller than normal, and its surface studded with miliary tubercles.

Gall-bladder measures from the neck to the fundus 8 inches. It extends 5 inches below the margin of the liver. It is pear-shaped, has thin walls, and is filled with a clear serum. Its outlet is occluded by what appears to be adhesive inflammation. There are no adhesions anywhere in the region of the liver, and no mass except the mere thickening, which is easily explained on the theory of some old, simple, inflammatory action.

The duodenum is thin. Stomach moderately dilated. No new growths.

This patient probably died from the effects of his nephritis.

Certainly, the gall-bladder lesions would not explain it, and the tuberculosis was not extensive enough to have caused death unless there were some brain-lesions. The brain was not examined. In all probability,

the man had passed a gall-stone at some remote period, and the inflammation was the result.

Discussion.—Dr. A. C. Abbott referred to a condition that occasionally occurs in animals under experimentation. In studying the properties of the typhoid organism it had been shown that, contrary to the opinion hitherto held, animals into which the bacterium coli commune was injected did not all die at once, but some lived quite a long time—as much as one or two months, or even longer. They showed very little evidence of disease, but rather a disturbance of nutrition, and lost weight gradually and regularly. The only pathologic lesions found were minute areas of necrosis in the liver and many might be seen upon the surface. There was generally a very greatly distended gall-bladder filled with clear bile, in which floated amorphous masses. The only species of bacteria found was the bacillus coli communis. The liver seemed to throw it off when it was injected into the circulation. This liver, with its dilated gall-bladder, strongly recalled the appearances seen in the experimental animals. It would be extremely interesting to know if the same condition existed here as in the case of animals, though the causation might have been quite different.

The President said: Occasionally cases of obstruction of the cystic duct are seen. In these instances the bile is absorbed, and the fluid which remains in the gall-bladder is simply a mucous secretion from the glands of the gall-bladder. Under such circumstances the tumor in the abdomen becomes enormous, and even simulates tumors of the lower half of the abdomen, such as the ovary. Cystic disease of the ovary and kidney and of other abdominal organs has been mistaken for an enlarged gall-bladder. He had seen several such cases; many can be seen in Blockley. If one wanted to write on the diseases of old age he would have to include large sections on diseases of the biliary passages. Many old people die of disease in this region, especially inflammation with perforation.

Dr. M. H. Fussell, in reply to Dr. Abbott, said that no bacteriologic examination had been made. He did not think it would have been much good, as the man had been dead forty-eight hours.

December 12, 1895.

7. *Carcinoma of the stomach.*

Exhibited by M. H. Fussell, M.D.

The patient was a painter, aged thirty-eight years, who two months ago began to have nausea, vomiting, and some flatulence. The pain was very severe, and came on after eating. The appetite was good; the bowels were constipated. His weight in five weeks had fallen from 215 to 163 pounds.

In the epigastrium was a well-marked, quite movable mass in the region of the pylorus. He was admitted to the wards of the University Hospital and placed on simple treatment. His stomach was much dilated and was washed out. The next day he was better, and had no pain. This was the first day he had been free from pain in two months. The next day also he was better. The third day he was very weak. The next day I received word I must come at once if I wanted to see him alive. He died half an hour after I got there. At the postmortem the stomach was found to be dilated. At the pyloric orifice there was a mass surrounding the entire valve. The pylorus admitted the middle finger.

Why this man should die it is difficult to say. There was no ulceration and no secondary growths. He probably died from toxemia due to the absorption of retained matter in the stomach. I can explain it in no other way. The stomach contained 600 c.c. of coffee-ground material.

December 12, 1895.

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8. *Perforating round ulcer of stomach.*

Exhibited by H. M. Fisher, M.D.

The patient from whom the specimen was taken, Charles L., aged thirty-eight years, single, born in Germany, was admitted to the medical ward of the Episcopal Hospital, November 20, 1895. Dates his present trouble from the passage of a tapeworm eighteen months ago. Since then his appetite has been failing, and he has suffered from dull pain, referred to the epigastrium immediately after eating. He has also been much constipated, for the relief of which symptoms he has resorted to the use of cathartics.

For three weeks before his admission to the hospital the above symptoms were aggravated, and he was not able to retain any solid food.

The pain was not constant, and he had vomited neither blood nor coffee-ground material.

Upon his admission to the hospital he was extremely emaciated, and his extremities were cold. No tumor could be made out. His heart and lungs were apparently normal.

Examination of the stomach-contents showed a diminution of the normal acidity.

As he could not retain solid food he was fed chiefly upon peptonized milk and preparations of beef. Vomiting was a frequent, but not a constant symptom. He often vomited peptonized milk about an hour and a half after taking it. His stools were normal in color until a few days before his death, when they became liquid and very offensive. Only once during his stay in the hospital was there vomiting of coffee-ground material. Efforts to feed him by nutrient enemata failed, as his rectum did not retain them. He became progressively weaker, and died January 1st.

At the autopsy the right lung was found to be tightly adherent at its apex, and there were two small cavities filled with pus at the apex, and there were several others near the base, containing greenish, offensive pus, but no tubercle granulations were found. The left lung was normal.

Heart small, but cavities and orifices normal.

The liver was small, hard, and had a waxy appearance. It was not examined for amyloid infiltration.

Adherent to the posterior wall of the stomach, near the pylorus, an abscess-cavity was found containing pus, measuring 15 by 3.5 cm., opening into the stomach cavity by an oval aperture, 8 by 3 mm. in size.

The pyloric end of the stomach was adherent to the right lobe of the liver, and the pylorus showed thickening of its walls, and stenosis. The edges of the ulcer were not indurated.

The kidneys were pale, and their cortex was reduced.

The other organs showed no changes of importance.

Discussion.—Dr. A. Stengel believed the specimen was one of gastric ulceration, with secondary abscess of the wall of the stomach, with subsequent perforation. The thought would arise, on looking at such a specimen, whether it began as a case of idiopathic abscess of the wall of the stomach or as a gastric ulcer. It is more likely to have begun as a gastric ulcer. The specimen was interesting from a clinical standpoint, because the disease had been diagnosed during life.

Dr. Fisher, in reply to Dr. Stewart, who asked how the dilatation was explained, said: As the abscess was close to the pylorus, there may have been some inflammatory thickening. *January* 9, 1896.

9. *Unusually large gallstones.*

Exhibited by J. McFarland, M.D.

I present these interesting gallstones through the courtesy of Dr. Mattern, the Coroner's physician.

Unfortunately there is almost nothing to say about them, the details of the case being simply as follows:

Mrs. K., aged sixty-nine years, was admitted to the Episcopal Hospital two weeks before death, having fallen down stairs and injured a knee. Up to the time of her death she had never suffered from any except slight ailments. She died, after having been in the hospital two weeks, from valvular disease of the heart and congestion of the kidneys.

The existence of these enormous calculi had never been suspected. The gall-bladder from which they were taken was normal, and there was no disease of the liver. It is interesting to note that such extensive calculi can form without inducing any symptoms.

Discussion.—Dr. Bolton asked if cultures had been made from the inside of the stone. The colon-bacillus had been found as a nucleus around which the stones had formed, and he desired to know whether it had been searched for.

Dr. R. H. Downes, in replying to Dr. McFarland's inquiry as to whether any one present had ever seen a larger series of gallstones taken

from one gall-bladder, said that a few years ago he had seen a woman who claimed to have passed a peck or more.

The President said he had never seen such an extraordinary specimen. He had removed, postmortem, a gallstone from the gall-bladder of a patient who had cancer of the gall-bladder and of the liver, and this stone was about two-thirds the length of the one shown by Dr. McFarland. The case was one of contracted gall-bladder, which had been the seat of carcinoma. At the time that he reported the case, with the measurements and weight, it was referred to by a number of journals as the largest on record. This series is extraordinary. He regretted that he could not bring forward some specimens that he had been collecting. The subject was very interesting, and he thought it might be well to bring up something of this character and discuss it in all its branches. He recalled an autopsy on one of his patients who had died from suppurative inflammation of the gall-bladder and gall-ducts and from obstructive jaundice. The common duct was the seat of obstruction, and the gall-bladder was filled with calculi, but was not unduly enlarged. Throughout the entire liver the ducts clear up to the surface were filled with gallstones. The surface of the liver was uneven, and this unevenness was due to collections, about one-quarter of an inch beneath the surface, of small gallstones. He could conceive of the patient passing an enormous quantity, but did not remember how large a peck would be. It showed, however, very clearly how useless it would have been to have operated, and especially according to the modern methods. There is no doubt that she would have passed stones innumerable, and so soon as the incision was closed the duct would be stopped up again.

Dr. E. B. Sangree asked whether the liver had been very spongy or not. He had removed a specimen very similar to that in which the common gall-duct was closed up, and a small stone appeared at its entrance into the intestine. The ducts in the liver were distended and filled with granular matter. The liver was almost lung-like in structure, and the microscope showed that the cell structure had nearly entirely disappeared and its place had been taken by fibrous tissue. The appearance was very peculiar.

Dr. Downes said that the peck he had referred to had been passed in the course of a year. The patient had lately informed him that she had been passing a material which she had never passed before, and which he thought was bile. She was getting very thin, and evidently had some trouble with her gall-bladder.

Dr. McFarland, in reply to Dr. Bolton's inquiry as to whether

cultures had been made from the inside of the stone, said he thought not. *January* 23, 1896.

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10. *Carcinoma of stomach; gastro-enterostomy by Murphy's button; death after four weeks.*

Reported by H. A. Hare, M.D., by courtesy of W. W. Keen, M.D.

John A. F., aged fifty-two years, of Clearfield, Pa., was sent to the Jefferson Hospital January 3, 1896, through the kindness of Dr. S. C. Stewart, of Clearfield. His father died of gangrene of the leg; his mother of paralysis at seventy. One uncle died of cancer of the liver; two cousins are said to have died of cancer of the stomach. There is also a history of tuberculosis in one uncle's family. One brother died of pneumonia, two brothers and sisters are living and in good health.

He had never been ill until two years ago, when he was successfully operated on for a hydrocele. About a year ago he had occasional attacks of nausea after eating. Last July he began to feel a soreness and tenderness at the pit of the stomach. This was increased by eating or driving in a wagon. About the end of August he began to lose his appetite and grew weaker, but he continued his work as a farmer until three weeks ago. During the last three weeks he has vomited repeatedly, some of the matters vomited being a dark coffee color. Vomiting relieved the soreness over the abdomen. He has lost fifty pounds in weight since last spring.

Status præsens. There is considerable increased resistance, but no distinct tumor perceptible in the region of the pylorus. The examination is not satisfactory, by reason of the tenseness of his abdominal wall. Inflation of the stomach by Seidlitz powder, the two portions being given separately, gave the same result and showed considerable dilatation of the stomach, especially in its lower left portion. A test-meal revealed no free HCl. The urine is acid, sp. gr. 1015; it contains neither sugar nor albumin. His temperature is 97.3°, his pulse 88, his appetite very poor, and no solid food is retained.

Operation, January 8, 1896: An incision in the middle line soon revealed the stomach, and an examination showed that the pylorus was the seat of a tumor moderately adherent to the liver and that the glands were involved to a considerable extent. Accordingly, I decided to do a gastro-enterostomy. The small intestine was very difficult to find, lying very low, so that even the Trendelenburg position did not

make it fall toward the opening sufficiently to catch it. Finally, by enlarging the opening, I passed in my hand, seized the intestine and stripped it first one way. In doing so, I found the wall was becoming progressively thinner and, therefore, I was tracing it toward the colon, whereupon I reversed the stripping and carried it upward until I came near to the duodenum. I then did a gastro-enterostomy, using Murphy's button, the one and a quarter inch size. As the opening did not seem to be quite closed, and I feared leakage from a little protrusion of the thickened mucous membrane, I carried a continuous right angled Cushing suture all around the button. He lost almost no blood, and the operation was quickly done, but in spite of that he suffered greatly from shock, his temperature dropping to 94.8° for several hours after the operation, but four hours later had risen to 99°. The highest point reached was on the sixth day, when it marked 100.2°, but by the next day had fallen to the normal, and so remained. In the week preceding his death he had gained one pound, and in the preceding week four pounds in weight. On the morning of February 5th, at 8 o'clock, he was suddenly seized with collapse, and died at 10 A.M. His weight the day before he died was eighty-nine pounds.

Postmortem was made by Dr. David Bevan, February 5, 1896.

External examination: Body greatly emaciated. Small bed-sores over left trochanter and left acromion process. Postmortem lividity seen only over the posterior aspect of trunk. Postmortem rigidity in the extremities only, and but to a slight degree. The muscles of the abdominal wall are greatly attenuated, dark-red in color, and quite tough. The subcutaneous fat has almost entirely disappeared from the abdominal wall. The incision made at the time of the operation in the abdominal wall has completely healed.

Abdominal cavity: Slight fibrinous adhesions extend between the abdominal wall about the line of incision and the omentum and intestines. Adhesions have also formed along the course of the round ligament, and also extend from the abdominal wall to the jejunum, duodenum, and pylorus, intimately binding together these structures. The cavity contains one quart of a turbid, opaque, greenish-yellow fluid, in which are a number of small fibrinous masses.

The liver extends well over into the left hypochondriac region; its color is dark chocolate. The gall-bladder protrudes one inch beyond the anterior margin of the liver; and it is greatly distended.

Firm fibrinous bands have developed along the whole extent of the greater curvature of the stomach, and, passing downward, have become attached to coils of the jejunum.

Thoracic cavity and pleural sacs: The lungs collapse upon opening the thoracic cavity.

The left pleural sac contains twelve fluidounces, and the right pleural sac two fluidounces of clear serous fluid.

Pericardium: The cavity contains one fluidounce of clear serous fluid. The membrane is apparently healthy.

Heart: The left ventricle is contracted. The right auricle is greatly dilated and contains mixed clots. The right ventricle contains a small currant-jelly clot and a small amount of fluid blood. The left auricle contains currant-jelly clots and a small quantity of fluid blood. The left ventricle contains but a very small quantity of fluid blood. The tricuspid valves, though thickened, are not contracted. The pulmonary and aortic valves are normal. At the base of the anterior leaflet of the mitral valves are several small atheromatous patches. The heart-muscle is dark red in color and quite tough. The aorta, pulmonary, and coronary arteries are atheromatous.

Lungs: The left lung is soft, and posteriorly it is the seat of marked hypostatic congestion. The anterior margin is emphysematous. On section, the lung is highly edematous, and the mucous membrane of the bronchial tubes is intensely red, thickened, and covered by a yellowish, tenacious mucus. In the apex of the right are several small, hard nodules which have the appearance of healed-in tubercular areas. Otherwise the lung presents the same appearance as does its fellow.

Spleen: The organ is very small, weighing not more than two ounces. The capsule is wrinkled and light blue in color. On section, the organ is quite firm and dark red in color. The Malpighian bodies and stroma stand out prominently.

Kidneys: The perinephric fat is completely absent. The left kidney—the capsule is edematous; the organ is lobulated and cuts with marked resistance. On section, the surface line of the cortex is slightly irregular. The cortex is very pale and striated. The capsule strips off readily, and the surface exposed is smooth and shining. The right kidney presents the same characters as does the left.

Numerous delicate fibrinous adhesions extend from the inferior surface of the liver to the head of the pancreas and lesser curvature of the stomach.

Intestines: Upon removing the intestines the Murphy's button is found in the ileum five feet from the ileocecal valve. It lies with its lumen transverse to the long axis of the intestine. For several inches about the position of the button the walls of the ileum are hyperemic, and there are several small areas of hemorrhage into the submucosa. The button is freely movable.

Stomach: The carcinoma involves the pyloric extremity of the stomach and the greater part of the duodenum. The pyloric orifice is slightly obstructed by the projection of a small mass of carcinomatous tissue. Organization at the seat of anastomosis between stomach and jejunum is well advanced.

Pancreas: Although this organ is involved in numerous fibrinous bands it appears normal.

Liver: The liver is very small, weighing not more than two pounds. The capsule is wrinkled. The organ cuts with marked resistance. It contains but little blood. Just beneath the capsule on the superior surface of the right lobe are several small greenish-colored cysts. They contain a transparent, amber-colored fluid of low specific gravity. The organ is very tough and fibrous.

The *brain* was examined, but no gross pathologic lesions were found.

Four ounces of fluid were removed from the abdominal cavity and placed in a glass vessel. When examined, ten hours later, a large, loose coagulum, in volume representing 50 per cent. of the whole, had formed. *February* 26, 1896.

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11. *Gastro-enterostomy for carcinoma of the stomach by Murphy's button; death in twenty-four hours.*

Exhibited by H. A. Hare, M.D.

The following notes are from Dr. Keen's records: Mrs. B. S., aged fifty-two years, was admitted to the Jefferson Hospital February 5, 1896, under the care of Prof. H. A. Hare. Her mother and father died of old age. Four sisters and a brother are living and well. She herself was always well till four years ago, when her menstruation ceased. Since then she has had prolapse of the uterus.

Her present trouble began four months ago, with anorexia, pain in the back and sides, of a very sharp, stabbing character, nearly constant, and at the same time she had difficulty in retaining any food. Everything was vomited immediately after eating, and with the food ejected there were mingled whitish stripes and sometimes black lumps looking like liver (blood-clots). The pain was relieved by the vomiting. During the past week she had pains in the region of the stomach. She had lost thirty pounds in the last four months; her bowels are constipated and the evacuations consist of black lumps (blood-clots). She has had much headache and is quite weak. Temperature 97.8°, pulse 88, respiration 26. Tongue coated, eyes sunken.

In order to relieve her from the distressing vomiting, and especially in view of the bleeding, which, as will be observed, took place by the mouth and the bowel, and with her entire consent and Prof. Hare's approval, I agreed to do a gastro-enterostomy with the object of giving her relief from the constant vomiting.

Operation, February 11, 1896: A vertical incision in the middle line instantly disclosed a stomach which was entirely diseased, excepting a portion at the cardiac extremity of the greater curvature. The disease was more extensive than the external examination showed, because it had extended along the gastrohepatic omentum to the liver without involving the gall-bladder, and there were also a number of small pin-head nodules on the abdominal wall where it had been in contact with the stomach, apparently arising by inoculation. As nothing else evidently could be done, I made an immediate gastro-enterostomy between the upper jejunum and the greater curvature of the stomach by means of a Murphy's button, using no sutures. The operation was quickly done, and she did not suffer much from shock, but simply died of the exhaustion of her disease about twenty-four hours after the operation.

Postmortem notes by Dr. Bevan:

External examination: Body fairly well nourished. Postmortem rigidity very slight. Small bruise, about one and one-half inches in diameter, just beneath the center of the right clavicle. Edema of the legs, most marked about the ankles. Considerable subcutaneous fat in abdominal wall. The muscular tissue is apparently in good condition. No fibrin between the edges of the incision, made at the time of operation, in the abdominal wall.

Abdominal cavity. Small quantity of clotted blood lying just beneath the line of incision in the abdominal wall. The tumor extends from the left hypochondriac region downward and inward to the lower part of the epigastric region. The upper border of tumor on a level with lower border of seventh rib.

Examination of organs. Spleen: Of about normal size. Capsule has a pale bluish color. On section, the organ is quite firm. The stroma is increased and the Malpighian bodies are prominent.

A large number of small carcinomatous nodules, varying in size from a small pea to a hazelnut, lie behind the pyloric extremity of the stomach, and by extension upward pass into the transverse fissure of the liver.

The gall-bladder is in no way involved.

Kidneys: These organs are about of normal size; their contour is

slightly irregular, and they are much firmer than normal organs. The capsules are slightly adherent.

The cortical substance is, over a few of the pyramids, slightly increased; while over others it is decreased. In color it is grayish, with marked red striations. The red striations are due to hyperemia. Blood drips quite freely from the cut surface.

The pyramids are of a dark-red color, with fine yellow lines radiating from apex toward the base. Small quantity of fat in pelvis. Several small cysts are to be seen in the cortex immediately beneath the capsule.

The stomach, duodenum, and upper portion of jejunum were removed, but no incision made into them. In the removal it was noticed that the loop of intestine between the pylorus and that portion of the jejunum attached to the stomach looped under the transverse colon.

The head of the pancreas is firmly adherent to the pyloric extremity of the stomach, and it is in close connection with the carcinomatous nodules previously mentioned.

The pancreas does not seem to be involved in the carcinomatous process. *February* 26, 1896.

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12. *Carcinoma of pancreas, with metastasis to stomach and liver.*

Exhibited by H. A. Hare, M.D.

The following is the history of the case as prepared by the resident physician and the postmortem notes of Dr. Williams:

H. F., aged forty-three years, married, occupation bartender, was admitted to the Jefferson Hospital February 26, 1896.

Family history. Father and mother both dead; cause and time unknown. No brothers; no sisters.

Personal history. Suffered from usual diseases of childhood. Twenty-two years ago he had a painful affection of right ankle, attended with swelling which extended to the knees. The trouble came on spontaneously, and was unattended by fever. He was confined to his bed for seven weeks, and wore a silicate dressing. Had gonorrhea eighteen years ago; at one time drank freely. Morning retching one year back.

Present attack. Occurred four weeks ago, with a sense of weight in the epigastrium and flatulence, which was relieved by vomiting; also had dull, heavy pain, which emesis did not relieve. Had no headache, but experienced drowsiness and weakness. Had some cough, accompanied by expectoration of a whitish material of thin consistency.

Never coughed up blood; bowels regular; lost some weight in the past month.

On admission, temperature 97°, pulse 94, respiration 22. Pain across back in region of kidneys, attended with sense of exhaustion; appetite good; bowels regular, in one instance bloody; tongue clean; face sallow. Pain is also felt in right shoulder. There is enlargement of inguinal and axillary glands.

The patient is emaciated, but, barring pain in the back, is feeling fairly well. He has not vomited since admission.

He was given a test-meal with the result that Gunzburg's test showed the presence of free hydrochloric acid, though in diminished quantity.

Urine, light yellow; specific gravity, 1025, very slightly acid, no albumin, no sugar, no bile. On standing it throws down phosphates. On January 12th, albumin was found in the urine.

During his whole illness the patient suffered but little pain. Morphine, one-eighth grain, by mouth three times a day being sufficient to allay all discomfort, till within three days of his death.

At the autopsy the following notes were made:

Patient greatly emaciated; weight ninety pounds. Superficial veins of abdomen distended and enlarged, superficial glands enlarged.

Peritoneal Cavity: Two and one-half pints of bile-stained fluid.

Pleuræ presented evidences of a former inflammation, the adhesions being so great as to tear diaphragm.

Pericardium: Slight evidences of former inflammation localized.

Heart: Right auricle contained postmortem clots; both ventricles distended, and left especially filled with fluid blood, while right ventricle contained chicken-fat clot.

Aortic leaflet fenestrated.

Lungs: Both lungs pushed up as far as the numerous pleural adhesions would allow. Both lungs were nodular and almost of a fibroid consistency, but presented no signs of suppuration. Resembled fibroid phthisis and caused knife to creak when passed through them, as if calcareous. There were cavities in both apices the size of a walnut,

Spleen: Normal in structure and in position.

Kidneys: Cystic dilatation of pelvis of left kidney; atrophy of parenchymatous portion; right kidney normal.

Liver enlarged and occupying the greater part of abdominal cavity. On the left it extends as far as spleen, and on the right as far down as anteriorsuperior iliac spine. It also forces the lungs well up into the chest, stretching the diaphragm as far as pleuritic adhesions would allow. The tissue is hard, presenting large, umbilicated, yellowish

nodules. Gall-bladder empty. *Pancreas:* There is a distinct cancerous growth involving the head of this organ. *Stomach* presents carcinomatous growths at both cardiac and pyloric ends. *The intestines* are free from the disease. *Bladder* normal.

February 26, 1896.

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13. *Meckel's diverticulum.*

Exhibited by M. H. Fussell, M.D.

The specimen was removed from a man, dead of acute tuberculosis, and had no part in the diseased condition. It is a perfectly healthy, though yet an abnormal part of the digestive tract.

The diverticulum springs from the lateral aspect of the lower part of the ileum, and is 9 cm. long, 7 cm. in circumference at its tip, and 9 cm. in circumference at its base. It has a distinct mesentery, which springs from the mesentery of the ileum, but is attached to it only at the base. The end is free and not attached to wall of abdomen. It can be easily seen how such a sac-like projection can be a cause of serious inflammation of the intestinal tract. In this particular instance obstruction was probably prevented by the lower end being much larger than the upper. It is, of course, well known that this curious anomaly is the remains of the omphalomesenteric duct, through which, in early embryonal life, the intestine connects with the yolk-sac.

In certain cases the end, which in this case is free, is connected to the belly-wall by the obliterated part of the duct. In this way a fertile source of intestinal obstruction is furnished.

It may also, when the usual cord is slightly pervious or when the gut is attached close to the abdominal wall, be a source of fecal fistula. I have notes of one such case in which the fistula discharged intermittingly, and in which the small cord was about the size of a goose-quill and six inches long.

March 26, 1896.

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14. *Leucin and tyrosin crystals in the urine in acute yellow atrophy of the liver.*

Exhibited by W. S. Newcomet, M.D.

Dr. Griffith asked me to examine this specimen of urine, stating that he thought the case from which it came died of acute yellow atrophy.

My report is as follows: It was cloudy, yellowish-red, with a yellow

sediment. When filtered the liquid was more of a red color, while the residue upon the filter and the froth were yellow. It had a peculiar odor, not urinous, but more that of liver. It had a specific gravity of 1018; the reaction was neutral. It contained 2.3 per cent. urea (estimated by the hypobromite method); albumin in about one-twelfth bulk. Gmelin's test for bile showed remarkably well, both as a contact-test and by dropping the acid upon the sediment.

Microscopically, it contained casts both granular and cellular, leukocytes, and epithelium of various kinds; some of these cells were deeply stained yellow, while others were not; this staining did not seem common to any one class of cells.

A number of small round bodies, some with thorns upon them, suggested urates of ammonia and soda, but were of a deeper red color; they looked like bile-acid crystals; this seemed perfectly consistent with the amount of bile present. Few other crystals, of no interest, and spermatozoa were also present.

Leucin and tyrosin being suspected, about half a cubic centimeter of urine that had been boiled and filtered was evaporated upon a glass-slide, by starting with two or three drops, and adding urine drop by drop until the above quantity was evaporated, then covered by a cover-glass and allowed to cool. The specimen contained numerous round bodies that looked like decolorized red corpuscles, both in size and refractive appearance; here and there was one that was larger and showed marked radiation. With these were numerous small crosses that looked like two phosphate crystals, but when focussed they could be seen to taper toward the middle; there were so many other crystals present that it made it somewhat doubtful if these crystals were leucin and tyrosin or not.

The following method yielded very good crystals, and enough for several chemic tests: About 10 c.c. of urine that had been boiled and filtered was treated with ammonium hydrate, then with a solution of basic acetate of lead; it was then filtered, and to the filtrate was added sulphuretted hydrogen to precipitate the excess of lead. It was again filtered, and the filtrate evaporated slowly. Passing the filtrate through animal charcoal improved the crystals.

To separate the leucin and tyrosin, it is best to recrystallize several times; the first is more soluble and remains in solution, while the latter crystallizes first.

Piria's test for tyrosin and Scherer's test for leucin, as was the test by sublimation for the latter, were readily obtained.

Now and then crystals resembling tyrosin are found in urine after the

administration of salol, and with these crystals are some spheres of urates of ammonia and soda in this specimen, and had not the patient from which this second urine came been in good health, leucin and tyrosin would have been suspected.

These crystals are probably salicylic acid or some derived product. One would be led to believe that they were a derived form, simply from the fact that they are so rarely seen.

Discussion.—Dr. Alfred Hand said the case reminded him of one he saw at the German Hospital under Dr. Wilson's care, with a very similar history. The patient was not so seriously ill, was jaundiced for some time, and had delirium and fever. The urine contained numerous casts, albumin, and leucin and tyrosin crystals. It was thought the case was one of acute yellow atrophy, but the man recovered. Possibly the case was one of Weil's disease or some infection affecting the liver.

Dr. Judson Daland said that urate spheres without spicules might be mistaken for leucin crystals.

Dr. A. E. Taylor said that seemingly considerable difficulty had been experienced in separating the crystals, and tests might have been made for lactic acid; in cases of acute yellow atrophy of the liver which have been examined for it, lactic acid had been found. It is not found in other conditions, except possibly in pernicious anemia. In addition to marked quantities of lactic acid, a large excess of ammonia had been found, and this was equally true in cases of chronic hepatic cirrhosis.

April 9, 1896.

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15. *Gallstones—peritonitis and death.*

Exhibited by M. H. Fussell, M.D.

The patient from whom the specimen was removed died just ten minutes before I entered the house to see her in consultation with Dr. A. H. Mellenk, of Roxborough. To that gentleman I owe the privilege of showing the specimen.

The history is as follows: Mrs. W., aged twenty-six years, had been subject to attacks of epigastric pain, sudden in onset, lasting a few hours and suddenly ending, ever since she was a child of fourteen. Until her fatal attack there was never jaundice.

On April 3d she was seized with severe pain, which was speedily relieved by a hypodermatic of morphine. April 4th she was comfortable,

but slightly jaundiced. April 5th there was a sudden exacerbation of the pain accompanied by uncontrollable vomiting, severe abdominal tenderness, and increasing jaundice; temperature 104°. She died at 9.20 on April 5th.

Postmortem *twenty minutes* after death. Body of a well-nourished woman; skin and mucous membranes deeply jaundiced. On cutting through the skin it bled freely from the veins.

The abdominal cavity alone was opened, and was quite as warm as in a living being.

The peritoneum was lusterless, not reddened, but covered in numerous places, especially where the omentum was in contact with the intestines, with flakes of recent lymph. The pyloric end of the stomach was tightly adherent to the tip of the gall-bladder, causing the stomach to be of hour-glass form.

The appendix was normal and lay back of the cecum.

The right kidney was tightly adherent to the under side of the liver near to the gall-bladder.

The duodenum was also adherent to the same region.

The liver was deeply congested. It was perfectly livid before any cut was made in its vessels. When the vessels were cut the viscus drained and became much paler.

In order better to dissect the parts the liver, stomach and duodenum, right kidney, pancreas, and spleen were all removed together. Stomach of normal size, but of hour-glass shape by reason of the adhesions above described. The mucous membrane was normal, no ulceration being seen upon its surface; the rugæ were marked. A gallstone was found in its pyloric end. The stone was about the size of a hazel-nut.

The duodenum was adherent to the liver. The papilla of the gall-duct was as large as a silver three-cent piece, and had an opening which easily admitted an ordinary pen-holder. The right kidney was normal, but so tightly adherent to the mass that the adhesions had to be severed by a knife.

The spleen was large and soft.

Pancreas normal.

Liver congested and its tissue deeply bile-stained.

There were a few patches of recent lymph over its surface.

Gall-bladder not distended; its surface almost covered by the adherent organs. The upper surface was tightly adherent to the liver. It contained a dram of bile and thirty-three gallstones. The stones varied in size from a hazel-nut to half a pea, were very white, looking much like clots of milk, and were marked by the typical facets.

The cystic duct was not dilated, and its outlet was partially occluded by a small stone. The common and hepatic ducts were about the same size, dilated to one-half inch when flattened.

The stones were soft, laminated, and microscopically consisted of a few cholesterin-plates, of degenerated epithelium, and bile-acids.

The cause of death, general peritonitis without rupture of the bladder or ducts, must be rare.

Unquestionably there was an exacerbation of the inflammation surrounding these parts, possibly a rupture of some of the old adhesions, and consequent peritonitis.

I am unable to find the conditions noted in any of the text-books; while they all, of course, note the local peritonitis and a general peritonitis dependent upon a rupture, they do not mention a condition similar to the one reported.

This postmortem was made with an old pocket bistoury, the only instrument used. The abdominal incision was closed with sewing-thread and a carpet-needle. *May* 14, 1896.

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16. *Stomach from a case of arsenic-poisoning.*

Exhibited by H. W. Cattell, M.D.

A. G., aged about fifty years, had been discharged from his position some ten days ago, and was naturally despondent in regard to his future means of support. A box of "rough-on-rats," which contains about 90 per cent. of arsenic, afforded the ready means of ending his life. You will notice on examination of his stomach, removed this morning, the characteristic appearance of marked hemorrhagic gastritis throughout the entire mucous membrane, and that petechial hemorrhages are found in great abundance, these being specially marked at the pyloric end of the stomach. A coagulation-necrosis is also visible.

The man had vomited a considerable amount of blood and mucus previous to his death, while at the autopsy there were about ten ounces of blood and mucus found in the stomach. There was some enteritis, the underclothing being stained with thin fecal matter containing a considerable amount of bile.

The specimen under the microscope shows octahedral crystals of arsenic prepared according to Reinsch's method. *May* 14, 1896.

17. *Series of stomachs from cases of progressive pernicious anemia.*

Exhibited by Alfred Stengel, M.D.

The following series of stomachs illustrates some of the conditions met with in cases of progressive pernicious anemia. It is not my present intention to enter upon any discussion of the relation of these pathologic conditions to the etiology of the disease. I have had occasion to enter upon this question more fully in other places.

The first authority who pointed out a relationship between pernicious anemia and gastric diseases was Fenwick.

1. This author described fatty degeneration of the tubules, and believed that this lesion was the cause of the disease. Subsequently a number of authorities referred to similar degeneration or to atrophy of the mucous membrane of the stomach or of the duodenum. In these cases the mucous membrane is thinned and smooth, the normal rugosities much reduced, the surface of the mucosa presenting none of the fine granulation visible in health. In all of the cases of this description that I have encountered there has been reactive hyperplasia of the connective tissue or of the mucosa itself or of the submucosa.

2. In a second group of cases there is marked glandular gastritis, the tubules being proliferated as in cases of hypertrophic inflammations of the mucous membranes, and the intertubular tissue markedly infiltrated with round cells and thickened by more or less connective-tissue hyperplasia. In some of these cases actual hypertrophy or pseudohypertrophy of the mucous membrane may cause projecting polypoid growths, as in an instance recorded below.

3. In a third group of cases the wall of the organ is thickened and the lumen encroached upon by the contraction of the organ and the hyperplasia of its walls. Of this form two striking examples are here recorded.

4. A fourth group of cases has been considered by some writers. In these, ulcerations of the stomach or of the intestine are discovered. I have no personal experience with such cases.

5. Carcinoma of the stomach has sometimes been described as associated with pernicious anemia. Such cases are referred to by Lubarsch and by older authorities.

My own case, referred to below, is an instance of this kind. In some respects it was not entirely typical. In particular, the condition of the bone-marrow deserves notice. This was only partially converted into the lymphoid form, and was rather firm. It must be recalled, however,

that conversion of the bone-marrow is not a necessary lesion of pernicious anemia any more than of other severe anemic conditions. Clinically, I have never found carcinoma presenting conditions of the blood found in this case, and should be at a loss to make any other diagnosis than that of pernicious anemia in this instance.

Case I.—H. B., a man, aged forty years, came under my observation several years ago, and was under my care at intervals until his death, a few months ago, when he died of increasing anemia. The patient had used alcohol rather freely, led a careless life, and had suffered with gastric symptoms the greater part of the last five years of his life. The anemia was profound, the number of corpuscles at one time being 600,000, and the quantity of hemoglobin 25 per cent. of the normal.

Postmortem, the stomach was found to be rather large, its capacity being about 2000 c.cm. The walls were thin and elastic, and the mucosa presented a rather yellowish and macerated appearance. Portions of the anterior wall were cut out for section, so as to avoid as far as possible the parts affected by postmortem digestion. Microscopically, the glands of these portions were found to be considerably and quite uniformly diseased. Portions placed in osmic acid exhibited the morbid changes particularly well. Almost all of the epithelial cells of the glands exhibited more or less fatty degeneration, and in some places the amount of fatty change was very great. The intertubular tissues were infiltrated with round cells, and the bloodvessels of the submucosa were decidedly thickened. The fibrous tissues were proliferated, and in the muscularis there was more or less cellular infiltration between the fibres.

Case II.—Mrs. B., a patient of Dr. Pepper, had been under treatment for chronic gastritis for fifteen years. She was more or less anemic during this time, but at the end of the period named the gastric symptoms and pain from the anemia grew steadily and rapidly worse. She was finally admitted to the hospital with marked symptoms and hematologic features of pernicious anemia. Hydrochloric acid was absent from the matters vomited, but no contents could be obtained with the stomach-tube on account of the patient's condition. A feeling of induration was made out in the epigastric space, but no distinct tumor was palpable. The blood, shortly before death, showed 900,000 corpuscles and 20 per cent. of hemoglobin.

Postmortem, the stomach was small and somewhat hour-glass in shape. The portion near the pylorus was contracted and cylindric, and the outer surface presented a rather sclerotic whiteness. On section, the wall was found increased in thickness, varying from 3 to 7 mm. The mucosa was rather hard and smooth, but finely granular. Micro-

scopically, the glands were preserved, as far as their number was concerned, if, indeed, there was not some unusual richness; but in shape the tubules varied greatly from the normal. They were nearly all compressed in portions, and, consequently, distorted, and sometimes this was remarkably evident. The cells of the glands were pale and granular, and to some extent atrophic. Unfortunately, no osmic-acid preparations were made. The intertubular tissues were hyperplastic and infiltrated. In places trabeculæ of fibrous tissue passed from the submucosa into the intertubular tissues of the mucosa, and in other places insular masses of dense sclerotic tissues pushed the tubules asunder. The bloodvessels were thickened. In the submucosa the fibrous-tissue overgrowth was more pronounced, and the bloodvessels were enormously thickened. Some of the vessels were almost obliterated by the hyperplastic thickening of their walls. The muscularis and serosa were similarly, but in comparison slightly, affected.

Case III.—Mrs. R., a patient of Dr. J. C. Wilson, was admitted to the German Hospital with the symptoms and general appearances of pernicious anemia. The examination of the blood showed a little over 1,000,000 red corpuscles and about 30 per cent. of hemoglobin. There were repeated vomiting and gastric distress.

Postmortem, the stomach was found a little smaller than normal, and, on section, the walls were thin. The mucosa was granular but soft, and at the fundus was seen a polypoid excrescence about 1½ cm. long, and of soft, papillomatous character. On microscopic section, this was found to consist of a rich accumulation of tubular gland structure, with round-cell collections between. The remaining parts of the stomach presented similar appearances. The glands were preserved, but the cells were granular and in many places wanting; and the interglandular tissues were densely filled with round cells.

Case IV.—Mr. S., a patient of Dr. Trau, was admitted to the German Hospital presenting the appearance of pernicious anemia. Examination of the blood showed a high grade of oligocythemia, with abundant nucleated red corpuscles and irregularities of size and shape. The progress of the case was that of progressive pernicious anemia. The patient occasionally vomited, but the vomita were never of distinctive character. No special examinations of the stomach-contents were undertaken.

Postmortem, a most pronounced degree of atrophic contraction was discovered. The stomach was reduced in diameter at its largest portion to somewhat less than the diameter of the large intestine in health. On transverse section the walls of the organ were seen to be enormously

thickened, the lumen being encroached upon so that at its greatest diameter it was not more than 3 cm. The mucosa, muscularis, and serosa were plainly distinguishable, and the former two were distinctly thickened. On microscopic examination the glands of the mucous membrane were found somewhat proliferated, the lining cells of normal appearance, and the lumen of the glands rather larger than normal. Between the tubules abundance of round cells was observable, and a groundwork of newly-proliferated connective tissue was more or less conspicuous in certain areas. The submucosa was considerably thicker than normal, and the bloodvessels of excessive caliber and surrounded by concentric layers of fibrous tissue. A few bloodvessels, almost completely obstructed by perivascular overgrowth, were discovered. The muscular wall of the organ was markedly excessive, and the nuclei more abundant than in the normal muscle-tissue. Intermuscular round-cell infiltration was apparent in certain areas. The serous coat was slightly hyperplastic.

Case V.—Mr. B., a patient in the wards of the German Hospital, presented the clinical appearance of pernicious anemia; the skin was of a lemon-yellow color, the tissues flabby; a tendency to slight edema of the ankles; marked weakness; palpitations and dyspnea pronounced; occasional vomiting. Indistinct resistance was encountered in the epigastrium, but a definite tumor could not be determined. Examination of the blood showed a marked reduction of red corpuscles (1,200,000) and about an equal reduction of the hemoglobin. The white corpuscles were a little in excess. The red cells were irregular in size and shape, and nucleated red corpuscles were present in some number, the smaller form (microblasts) being particularly abundant. Differential counting of the leukocytes showed excess of polymorphous elements. The patient gradually declined in strength and died of exhaustion.

Postmortem, a carcinoma was discovered on the posterior wall at the pylorus and invading the head of the pancreas and the under surface of the liver. The stomach was moderately distended and the pylorus little encroached upon. The mucous membrane of the body of the organ was practically normal in appearance. Microscopically, the tumor was found to be a carcinoma, but invasion had been sharply circumscribed.

Case VI.—Mrs. O., presented the clinical features of pernicious anemia. The blood-count showed 1,000,000 red corpuscles, 22 per cent. hemoglobin, slight leukocytosis, nucleated red corpuscles, and irregular forms of erythrocytes. Gastric symptoms were noted, but were never pronounced.

Postmortem, the stomach was greatly reduced in size, and, like Case IV., had an elongated, intestine-like structure. The greatest diameter

was about 4 or 5 cm., the lumen being narrowed by encroachment of the walls. Microscopically, the mucous membrane was proliferated. The glandular tubules were widened and more abundant than normal. The epithelial lining, however, showed no abnormality. The submucosa was proliferated and the vascular supply excessive. The mucosa was greatly thickened, the nuclei of the cells more abundant than normal, and occasionally round-cell infiltration between the fibers was observed. The serous coat was about normal.

Case VII.—Laborer, aged thirty-four years, with a tuberculous family history. Had a peculiar pigmentation of the hands, face, and forearm; a mitral systolic murmur; enlarged lymphatic glands in the groin, and some nodules in the abdominal wall, probably enlarged glands. The urine was light in color; hemoglobin at first 16 per cent., sinking gradually to 12 per cent. Red corpuscles sank from 1,500,000 at first to 500,000 at the last count. White corpuscles were also reduced in number. The red cells showed the usual changes of shape and size, with presence of nucleated cells, seen in pernicious anemia. At the autopsy the heart was found to be fatty, but the valves were healthy. The stomach was contracted to a very small size. The mucous membrane was smooth and glazed; the rugæ flattened, except near the pylorus. The mucous membrane and the muscular coats were greatly thickened in the form of a firm ring near the pylorus. The upper part of the small intestine resembled the pyloric end of the stomach. The gut was contracted and hard. The liver was of a uniform yellowish appearance and showed small hemorrhages near the cortex. The kidneys were pale, but otherwise normal. *June* 11, 1896.

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18. *Cirrhosis of liver in an infant.*

Exhibited by A. Hand, Jr., M.D.

The slide which I have placed under the microscope is from the liver of an infant, nine months old at death, and shows an extreme overgrowth of the perilobular connective tissue, with marked proliferation of bile-ducts. The microscopic view suggests hypertrophic cirrhosis, and at one time during the course of the case the edge of the liver could be felt near the iliac crest. At the postmortem examination emaciation, jaundice, and ascites were marked features, and the liver was shrunken so that the edge was above the costal margin, the organ

weighing 225 grammes (7½ ounces) and measuring 14 cm. laterally, 8¼ cm. antero-posteriorly, and 3½ cm. vertically. A scar at the lower border of the right kidney, and a thickened splenic capsule, strengthened the belief that syphilis was the causal factor.

I am indebted to Dr. Starr for the opportunity of making the examination. *June* 25, 1896.

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19. *Endothelioma of the omentum.*

Exhibited by Joseph McFarland, M.D.

Mrs. E. R., aged seventy-seven years, widow, mother of several children, scrub-woman by occupation, has always enjoyed unusually vigorous health, and been able to do a hard day's work, until the last illness became so severe as to confine her to bed a few months ago. No careful detailed history of her life was taken, but from my own personal knowledge of her she has been temperate and generally in good health. During the smaller epidemic of influenza in 1891 she suffered a bad attack.

The history of the present illness dates from March of the present year, when the patient lifted a heavy object and heard and felt a sharp crack in the abdomen. This was followed by gradually increasing pain and tenderness until the symptoms resembled acute peritonitis.

During the time that she was under my observation the acute phenomena passed away somewhat, and the symptoms became more like those of chronic peritonitis. Much flatulence was experienced. The stomach was retentive, and the bowel movements few in number and small in amount. The patient, however, had no appetite.

I confess that as the patient was ill and depressed, and had not shown evidences of disease other than abdominal, I made a most superficial examination of other parts of the body, and can report that careful physical examination of the abdomen revealed nothing in the way of a tumor or enlargement. There were no hernias. At one time I almost rejected the idea of organic disease and attributed the symptoms to colic. The condition, however, remained stationary, the woman suffered from much colicky pain, especially at night, and was only relieved when the almost constant constipation was overcome by enema or by purgatives.

The patient passed out of my hands into the care of several different

physicians. Dr. Marie Formad, who once attended her, tells me that she tapped the abdomen twice, both times withdrawing considerable fluid (I think once four and once six quarts). The disease ultimately became chronic and serious, and the patient was bedfast for three months before death. Toward the last no nourishment of any kind could be tolerated, and from the empty condition of the contracted stomach, as well as the extreme emaciation, it is not impossible that the patient starved to death.

Autopsy, September 8, 1896, twelve hours after death. Body embalmed with some preparation of formalin. Patient extremely emaciated. Upon opening the abdomen it was at once discovered that the abdominal viscera were badly diseased. There were a few adhesions between the organs and the parietal peritoneum of the anterior wall. When these were cut and the parietes turned back, the peritoneum was found to be thickened enormously, very rough, strewn over with pin-head and pea-sized nodules of white color, and everywhere intimately adherent except upon the surface of a large irregular tumor in the center of the abdomen.

It at once became apparent that the usual method of proceeding in making an autopsy must be abandoned, and the case studied as was possible.

Thorax: When the thorax was opened the lungs were found to be large, inflated, and normal in appearance, the heart completely hidden. The pleuræ contained some fluid, presumably embalming fluid.

Pericardium normal. Heart: Small in size, not as large as my fist; the surface marked by very tortuous atheromatous vessels. The tricuspid and pulmonary valves were slightly fibroid. The mitral valves were very much thickened, and somewhat curled; no calcareous deposits. The aortic valves were intensely diseased. The alteration in shape was very slight, but they were so calcified that they were no longer flexible, and they formed immovable impediments to the escape of blood, explaining the extremely loud aortic obstructive murmur which could be heard in the small arteries during life.

Abdomen: The chief object of interest centered about a large, flat, central mass just below the diaphragm, intimately attached to it and to the liver, and supposedly embracing the spleen. It was whitish-mottled in color, considerably larger than the hand, and smooth where free of adhesions. This mass was subsequently shown to be omentum. To its substance at the extreme left the spleen had been fused. The stomach was found, after considerable search, above and behind the omental tumor, imbedded in a general mass made up of transverse colon

and tumor-masses. The stomach was small and contracted, and showed no other signs of disease upon its mucosa than three suspicious ulcers (?), which may have been the cause of the vomiting. The pylorus admitted the little finger, and was not involved in the general mass, its structure free from disease. The esophageal opening was likewise normal. Liver: The liver was large, its surface covered by the thick capsule of chronic perihepatitis, and very adherent—sometimes by bands as thick as the little finger, but friable in nature—to the diaphragm, to the intestines, and to the omental tumor. Upon section, the structure of the liver was nutmeg from increase of fibrous tissue, but otherwise normal. The small intestines were partly adherent to each other, and were upon their surfaces, as well as upon the mesentery, studded with the miliary nodules already mentioned.

Pancreas: Normal. Spleen embodied in tumor-mass, atrophic, fibroid, and infarcted. The kidneys were a little larger than normal, were partially lobulated, and exceedingly hard. The capsule was somewhat adherent, and beneath it there were many small cysts. Upon section, the substance was pale, the little vessels gaped, and the appearances were those of arteriosclerosis. The right ureter was obstructed by implication with the pelvic mass.

The pelvic organs formed a solid mass of almost cartilaginous consistence, which was removed with great difficulty. The bladder was found to be free of disease—inside. The vagina, when explored by the finger, was normal, and the cervix normal; the os small, regular, and contracted. A probe introduced into the os found its way into a smooth cavity, of about the normal size of the uterine canal, exposed in a vertical section. The ovaries could not be found. Douglas' pouch was completely filled with a prolapsed mass of gut—supposed to be the sigmoid flexure—so intimately incorporated in the tumor-mass that it is only in section that they can be recognized as gut.

It must be conceded that in a case where the organs were so massed together as this the hope of carefully investigating each one is very small. What examination of organs could be made revealed one of those puzzling conditions in which a large tumor, having disseminated itself widely, is with difficulty referable to any particular primary source.

It did not arise in the stomach, in the liver, in the spleen, in the pancreas, or kidneys. The thoracic organs were, of course, out of the question. The remaining possibilities were genital organs, intestines, and the peritoneum—*i. e.*, the omentum.

Subsequent microscopic study of the tissues may throw some light

upon the origin of the case, but at present we must say that while the external margin of the uterus could be traced in transverse sections of the pelvic mass, it was impossible to say whether the disease beginning in it might not have spread to the adhesions and neighboring organs. Again, the intimate association of tumor-mass and sigmoid in the pelvis and transverse colon and omental mass precludes the possibility of asserting that they are not the starting-points. Perhaps microscopic study, by showing the type of the cells, will make these things clear.

Upon section the tumor-masses resemble carcinoma. They are grayish in color, granular, mottled with yellowish patches where fat is present; and here and there softened by myxomatous change, and fulfil all the descriptions of cancer.

From the general distribution of the growth, and especially from the manner in which it involved the omentum, I am strongly reminded of a similar case in the practice of Dr. Samuel Morton, which I saw through his kindness, in which an almost identical condition was present, and in which the microscopic examination showed the tumor to be an *endothelial growth.* *September* 10, 1896.

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20. *The lymphatic and epithelial structures of the fetal appendix vermiformis.*

Exhibited by A. E. Taylor, M.D.

The rich lymphatic tissue of the gastro-intestinal tract has long been the object of careful studies. I believe that it is to-day justly considered as a part of the lymphatic system, ranking with the spleen, marrow, and lymph-glands. That it is not a reaction to bacterial processes within the intestinal tract is made probable by the fact that it is well developd by the fifth month of fetal life. Schmidt, of Bonn, has studied the fetal intestinal tract, and by means of an elective mucin-stain (the Ehrlich triple mixture) has shown, furthermore, that the intestinal epithelium functionates as early as the fifth fetal month. I have repeated these studies upon three fetal appendices, aged respectively five, six, and seven months, sections of which I herewith present for inspection. In all three specimens the epithelial cells are seen to contain mucin (stained blue-green, while the protoplasm of the resting cells is a deep-red); that is, they are engaged in active secretion. This is not marked in the earliest case, but is well-marked in the other two.

The lymphatic tissue is very extensive, in some places comprising one-half of the wall, a solid mass of uninuclear, nongranulated lymphatic cells. What bearing these facts may have upon the question of the function of the appendix is outside our province here.

September 10, 1896.

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21. *Obliteration of gall-bladder.*

Exhibited by J. D. Steele, M.D.

The specimen shown was removed from a woman, aged sixty-five years. The liver is large and flabby, with an elongated right lobe. The organ is of a diffuse yellow color, and has the appearance of being in an advanced stage of fatty degeneration. The gall-bladder is thickened into a cartilaginous pouch, full of gallstones, and containing no bile. It is evidently an old condition caused by excessive formation of stones and inflammatory action, with contraction of the connective tissue of the bladder-wall. The cystic duct appears to be closed by the growth of the connective tissue at its point of entrance in the gall-bladder. In other respects the ducts are normal. There is a slight interstitial nephritis and chronic fibrous myocarditis.

September 24, 1896.

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22. *Internal strangulated hernia of the small intestine through the omentum.*

Exhibited by H. W. Cattell, M.D.

Dr. C. Y. White, resident physician at the Philadelphia Hospital, kindly furnished me with the following clinical notes of the case of W. M., a German, male, aged fifty-six years: The patient was a sign-painter by trade. He has had several attacks of lead-colic; uses alcohol moderately. He admits sexual excesses, but denies venereal history; has had chills and fever for two weeks. Twenty-two hours before admission he mixed chrome-yellow and Prussian blue paints. After mixing these colors he ate a meal from the table on which some of the dry powder remained, and he thinks some of the chrome-yellow might have been swallowed with his bread. Twelve hours after his meal he

was taken with severe abdominal pains, nausea, and a profuse cold sweat. This continued until his entrance to the hospital. On admission, temperature 97.3° ; pulse, 120 ; respiration, 26. Cold, clammy sweat ; distended abdomen, with extreme tenderness, especially marked in region of umbilicus and over the stomach ; nausea ; dry, coated tongue ; constipation ; scanty urine. After a glycerin and magnesium sulphate enema he had a stool of about a pint of nearly pure blood. He then grew progressively worse. The pulse was rapid and wiry ; several times he almost went into collapse ; vomiting was continuous. His urine contained fat-globules, fatty casts, fatty epithelium ; a trace of albumin. Just before his death, which occurred in collapse, the stomach-tube was passed.

Postmortem notes : The amount of rigor mortis is so very intense that the head is supported in the air by means of the tissues of the shoulder and neck. The man is very anemic and somewhat emaciated. On opening the abdominal cavity about a pint of fluid blood was found. The stomach, transverse colon, and duodenum are matted together where they come in contact by means of recent reddish fibrinous bands, which are apparently not due to bacterial infection, but merely to the separation of fibrin from the blood from a strangulated area of the small intestine. There is an opening in the omentum through which ten or twelve feet of the small intestine, with its contained mesentery, have passed. It is surprising to note how strong the constricting neck of the hernia really is, it being difficult to introduce the little finger between the omentum, which does not seem to be inflamed, and the bowels. The intestine and mesenteric attachment are dark-red and slate-black in color, and swollen. The intestinal wall is thickened ; the contents are dark-colored, malodorous, fluid, and stained with blood. The ileocecal valve is about three inches from the end of the constricted portion, free ; the portion of the ileum immediately above the constricted part is filled with fluid. Fatty degeneration is plainly to be seen in the heart, liver, and kidneys ; in the latter organ there is an acute parenchymatous nephritis on top of a chronic parenchymatous nephritis, with interstitial changes. No attempt was made to diagnose the presence of lead in the organs, but the history was undoubtedly that of lead-poisoning, and it would seem that the hernial protrusion had been caused probably by the violent peristaltic movements produced by this chemical substance. It would have been interesting, also, to have tested for increased toxicity of the colon bacillus, as a number of experimenters have recently shown that in artificially produced strangulation of the bowel this organism is increased in virulence. *September* 24, 1896.

23. *Glandular carcinoma of stomach and peritoneum.*

Exhibited by J. D. Steele, M.D.

The patient, a woman, aged sixty-five years, white, was admitted to the West Philadelphia Hospital for Women in the summer of 1896, suffering from a rapidly-growing tumor in her abdomen. Her condition was not such as to permit of an operation, and she died within a few hours after admission.

Autopsy. The abdominal cavity is much distended by fluid, which has a reddish tinge. The omentum covered the whole of the intestines, projecting down and touching the bladder. It averages 7 cm. in thickness. Anteriorly and laterally it is adherent to the parietal layer of the peritoneum. On section it is found to be extensively infiltrated by a new growth. It is soft and homogeneous, very friable, and of a pinkish-white color. The parietal layer of the peritoneum is covered with the same kind of growth over the surface of the abdominal parietes and intestines, the abdominal parietes, liver, and spleen. The epiploic appendices of the sigmoid are much enlarged. The peritoneum of the small intestines is almost unaffected. The omentum is firmly adherent to the wall of the stomach over the whole surface. They cannot be easily torn apart.

The stomach is small, and measures 18 cm. in its greatest length. It is closely adherent to the omentum on its whole anterior surface. On section its anterior wall is much infiltrated, and measures 20 mm. in thickness. The growth is of the same nature as that in the omentum. The surface of the growth is ulcerated in irregular areas, and seems, to the naked eye, to have undergone mucoid change. The esophageal opening and the pylorus appear perfectly normal. The posterior wall is not infiltrated except in its lower portion. The whole organ is very much contracted and the lumen is much diminished. The other organs are not affected by the growth, and appear to be fairly healthy.

Microscopic examination of pieces taken from the stomach and omentum shows the growth to be typical glandular cancer with considerable mucoid change.

The case seems to me to be of interest on account of the unusual point of origin for gastric cancer. The primary seat must be considered to be in the peptic glands, for the cells are cuboidal and typical of glandular cancer. Strange to say, the liver was not affected.

October 22, 1896.

24. *Tumor of gastro-intestinal tract and omentum.*

Exhibited by A. E. Taylor, M.D.

These enormous specimens were removed from a man, aged fifty years, whom they had choked to death. The duration of the case from the first symptoms was about eight months. He underwent an exploratory laparotomy, at which time quarts of gelatinous exudate were removed from his peritoneal cavity. Macroscopically the appearances are those of the so-called colloid cancer. It involves the stomach, the duodenum, the central portion and lower end of the ileum, the cecum, the left and right colonic flexures, the sigmoid and rectum, the omentum, and there are nodules all over the peritoneum. I have made a large number of sections from the involved portions of the stomach and intestines, but can find no site of origin; in none of the sections can the tumor be seen to extend through the tunica muscularis, and in all the sections the tunica mucosa is normal. The point of origin, therefore, remains entirely obscure. There are very few cells in the tumor, and most of these are not of definite type; it were difficult to say whether they are epithelial or endothelial. In a few sections, however, I have found cells which give the mucin-reaction of Schmidt, which I believe excludes an endothelial origin.

Discussion.—Dr. M. V. Ball recalled a similar case from Dr. Wolf's service in the German Hospital. It was considered an instance of peritonitis deformans, but on section presented an appearance similar to Dr. Taylor's case.

Dr. David Riesman had observed a case that presented itself like a case of plastic peritonitis of peculiar form—the intestines were bound together and covered over by a smooth, thick, opaque sheet of fibrous tissue, looking like icing, and reminding one of the icing-liver (Zucker gussleber) of the Germans. In cases where no primary growth is discoverable, the condition is generally a diffuse endothelioma; in his own case the intestine was the seat of carcinomatous growth inside of the thickened peritoneum.

Dr. Alfred Stengel had seen a secondary tumor somewhat similar to this one; the primary growth was in the rectum. There was a flat deposit in the diaphragm and liver, and the intestines were matted together. He had seen one case of primary endothelioma of the peritoneum, and one of the pleura a few years ago, the latter being peculiar

in that it had columnar cells. It was unquestionably endothelial, as it originated from the pleura. He believed that endothelial and epithelial cells were very similar; at times it was impossible to tell whether cells were mesoblastic or epiblastic or hypoblastic. *October* 22, 1896.

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25. *Primary cancer of head of pancreas.*

Exhibited by ALFRED STENGEL, M.D.

A primary cancer in the head of the pancreas, with occlusion of the common duct. The duct as well as the gall-bladder is enormously dilated; the latter contains many stones. The growth has ulcerated into the duodenum just below the pylorus. There are no metastases.

October 22, 1896.

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26. *Dilated stomach from pyloric obstruction; contracted kidneys.*

Exhibited by S. M. HAMILL, M.D.

The patient from whom these specimens were recovered was a woman, aged twenty-five years. Her father, who is living and well, had a sore on his penis prior to his marriage. There were never any secondary manifestations. The mother had one miscarriage and four healthy children; two of them are dead, one of measles, one of diphtheria. A brother, aged twelve years, is living and well. The mother died of some vague brain condition, and the physician in charge considered it probably of syphilitic origin. The subject in question had had measles and scarlatina in early childhood.

Since twelve years of age she has suffered, at various intervals, from nausea and vomiting, and, sometimes, had severe epigastric and lumbar pain. For some years the daily amount of urine voided was small. The result of many examinations revealed a persistent low specific gravity, with from a trace to a decided ring of albumin and a few pale granular and hyaline casts.

On June 12, 1895, following a period of twenty-four hours in which she had voided but a few ounces of urine and vomited profusely, the vomited matter containing a considerable quantity of blood, she suddenly developed a tetanoid condition, which involved all the muscles of

the body. This attack lasted from five to ten minutes. It was succeeded by a number of others, occurring at short intervals. The skin was dry and hot, temperature 101° F., and the arterial tension was very high. The abdomen was distended and tympanitic. It was noted, as the patient was under daily observation, that the attacks of vomiting occurred at intervals of a few days, and that the quantity vomited was profuse. As the quantity of food given was increased the quantity of matter vomited seemed correspondingly increased. In the course of the summer the patient had a number of attacks similar to the above. Toward autumn her condition improved, and by the beginning of the present year the attacks of vomiting had ceased entirely, and the patient was able to take solid food without discomfort.

The abdomen, which had been greatly distended and tympanitic, became much relaxed. This was so decided that, notwithstanding a rapid increase in weight, the clothing had to be persistently taken in at the waist. This improvement is especially emphasized for the reason that, in the light of the postmortem findings, it seems almost inexplicable.

During my absence from town, in the month of August, Dr. Stengel was called to see the patient, and found her in an attack of the same character as those previously described.

She improved, and was about again for some weeks, but the paroxysms of vomiting had recurred and were becoming more frequent; there was a rapid persistent failure in health, the abdominal distention and tympany was extensive, and the patient died on October 6th, after a series of violent tetanic spasms.

There was no urine voided during the last twenty-eight hours of life; only two pints in the last seventy-two hours. An analysis made ten days prior to death revealed a very faint cloud on boiling, and no casts.

The question as to the origin of the toxemia, whether renal or gastric, is interesting and difficult of determination. Either the renal or gastric postmortem findings would satisfactorily account for the complex of symptoms.

The autopsy was made sixteen hours after death. Rigor mortis was marked. Emaciation was extreme. The abdomen was very prominent and tympanitic throughout.

On opening the abdominal wall the stomach was found to be almost the only organ visible. There were three short coils of small intestine to be seen in the left iliac region. The pylorus extended below the right anterior iliac spine and pointed posteriorly. The cardiac end was concealed beneath the ribs, and on their removal was found to extend as high as the upper border of the fourth rib. There was a small area of

pericardium, about the size of a five-cent piece, visible at about the left sternal border opposite the fourth rib.

The esophagus and duodenum having been ligated, the stomach was removed and measured, with the following results: Greatest length 38 cm.; greatest diameter 12½ cm.; greatest circumference 37 cm.; pyloric circumference 16½ cm. It will be observed that these measurements show an extremely long stomach with a relatively small diameter, a condition which was much more evident when the stomach was *in situ* than at present. At the junction of the pylorus with the duodenum was found an extreme constriction, reducing the calibre to a diameter less than that of the little finger. It had much the appearance of having been encircled by a ligature. Immediately on either side of the point of constriction there was decided hardness to the touch. The stomach wall was thickened almost throughout, gradually fading as the cardiac end was approached, and increasing to the point of obstruction, near which point the hypertrophy became extreme. The upper portion of the small intestine seemed about of normal calibre, the lower portion was under the normal, and the large intestine, beyond the cecum, was extremely small and entirely empty. On opening the stomach the cause of the obstruction was found to be a large cicatrized chronic ulcer. There were several other cicatrices, all in the pylorus and in the duodenum; about two inches beyond the constriction was found an ulcer of more recent occurrence. The stomach contained about a half-pint of a brownish-colored liquid almost odorless. The inner surface was smooth and of a leathery appearance.

The kidneys were very small and of firm consistence. The capsule was adherent; the surface was granular, and on section the cortex was from two to three mm. in thickness. The suprarenal seemed enlarged. The liver was congested and, on section, had the appearance of being somewhat fatty. The spleen was rather hard. There was a small cyst of the left ovary. The lungs were free. The heart was very small, a fact that is noteworthy in view of the kidney condition.

Discussion.—Dr. Hare did not wish to discuss the pathologic side of the case, but desired to point out that, clinically, it was well recognized that dilatation of the stomach favored tetany; so did also other nutritive conditions.

Dr. Riesman thought that there had been a duodenal ulcer, as the remains could yet be seen. There were two points in the case that were of special interest: First, the sex of the patient. Perry and Shaw in their researches had found duodenal ulcers about three times as frequent

in men as in women. Second, the existence of Bright's disease in this case. Perry and Shaw had paid attention to the association of nephritis and duodenal ulcer, and had found a greater proportion of instances of Bright's disease in duodenal ulcer than could have been expected from the known frequency of the former disease. It is not impossible that the Bright's disease in this case played a *rôle* in the causation of the ulcer. *November* 12, 1896.

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27. *A case of fatal purulent peritonitis from perforation of a single ulcer of the ileum.*

Exhibited by Joseph McFarland, M.D.

The patient was found dead in an empty house; the coroner's jury rendered a verdict of death from alcoholism (?).

Autopsy. Body of an average-size colored man with well-developed muscles, but very little fat. No signs of external injury were observed. Rigor mortis was absent. From the mouth a greenish-fluid escaped. The patient had evidently been dead for some time.

Abdomen: Upon opening the abdominal cavity acute purulent peritonitis was apparent. The peritoneal cavity contained a large amount of greenish pus; the omentum, intestines, and, indeed, all the organs were united by soft, fresh adhesions, and the liver received an investment of fibrinous deposit that formed a true pseudomembrane covering the entire organ. The appendix was at once investigated as the cause of the condition, but, while slightly enlarged and injected, was not diseased. *Thorax:* The *pericardium* contained a normal amount of slightly purulent fluid. The *heart* was normal in every particular. The *pleura* contained a slight amount of clouded fluid. The *lungs* showed marked hypostatic congestion; there were old pleuritic adhesions at the left apex posteriorly, but careful inspection of the substance of the lungs failed to show any disease. *Abdominal organs:* The *spleen* was adherent to all the surrounding tissues by fresh, soft adhesions. It was about one and one-half times the normal size; the parenchyma soft, presumably from decomposition. The *kidneys* were slightly increased in size from cloudy swelling, but were otherwise normal. The *pelvic organs* were examined, and exhibited no pathologic change. The *duodenum* and *stomach* showed softening of the mucous membrane from decomposition. At the cardiac end of the stomach there was an annular blackish discoloration about four inches in diameter, evidently corresponding to the

distribution of veins containing decomposed blood. There were no ulcerations. The bile-ducts were patulous; the gall-bladder normal. The *liver* was discolored, blackish upon the surface; this discoloration, evidently due to decomposition, penetrating to a depth of half an inch, below which the substance of the organ presented no apparent changes. The liver was enlarged. The *intestine* now became the point of interest and was carefully examined. At the right iliac fossa there were adhesions extending between the cecum and surrounding parts, but when these were removed no lesion was found to exist apart from the general disturbance.

The intestine was carefully examined from end to end, and revealed but two small lesions, one an erosion of the mucous membrane, about the size of half a pea, appearing to be punched out of the mucosa; the edges were not infiltrated. The second was an ulceration, about the size of a Lima bean, with thickened edges, terraced center, and a central perforation, perhaps one-eighth inch in diameter. These two ulcers were about six inches apart, and were situated in the ileum about a foot above the ileocecal valve. The peritonitis was, no doubt, due to the escape of fecal matter from the perforation. The pus should have been examined bacteriologically, but the man had been dead so long and the tissues were so decomposed that no attempt to do this was considered to be worth while.

The possibilities of the case were numerous.

1. *Typhoid fever.* Cases are on record in which one or two ulcers were all that existed to indicate this disease. However, in the case under consideration there was no enlargement of Peyer's patches or solitary glands elsewhere. The mesenteric glands were not more swollen than might be expected in peritonitis; the enlargement of the spleen was scarcely sufficient to indicate typhoid. 2. *Tuberculosis.* Tuberculous ulcers of the intestine are generally secondary in adults to tuberculosis elsewhere. This man had no tubercles elsewhere; at least, none were discovered. Tuberculous ulcers are generally irregular, characterized by outlying miliary tubercles along the lines of the neighboring lymphatics, and only rarely perforate, while this case was without these characters. 3. *Embolism,* with resulting anemic necrosis, is generally an outcome of endocarditis. In this case there were neither other infarcts nor any lesions of the heart. 4. *Syphilis.* Syphilitic ulcerations of the intestine are rare. Like most specific ulcerations they are usually characterized by marked induration of the surrounding tissues. They are rarely single, do not generally perforate, and should be accompanied by signs of syphilis: stellate scars of the organs, gummata, changes in the blood-

vessels, etc., elsewhere; none of the conditions mentioned existed in this case. 5. Foreign bodies sometimes ulcerate through the intestinal walls, but in this case no foreign body was encountered. 6. Intestinal stones or masses of inspissated fecal matter have been known to erode the walls of the intestine and perforate. In this case, however, the intestine was empty but for a small amount of semi-fluid contents. 7. Parasites, such as round worms, occasionally perforate the walls of the intestine and bring about peritonitis. Here, however, there were no parasites either within the intestine or within the abdominal cavity. Moreover, the ulcer has every appearance of having existed for some time. 8. Septic ulcers also occasionally occur in dysentery, but this can at once be excluded by the absence of dysentery and the single lesion.

The examination of the ulcer itself, macroscopically, gives very little information about its nature. It is circumscribed by a rounded, infiltrated, but not cicatricial, border. Its surface is comparatively smooth, except at the borders, where there are a few slight, whitish, papillary projections, somewhat resembling tubercles, but, in my judgment, more likely fragments of seminecrotic character projecting from the more healthy tissue. The serous surface is smooth, except for a partial fresh adhesion, and shows no pathologic change except the rounded opening at the centre of the ulcer.

The microscopic examination is reserved, as I did not care to spoil the appearance of the specimen until it had been exhibited to the Society.

Discussion.—Dr. Swan had seen a similar case. A woman died after a kick in the abdomen. At the autopsy a round, perforated ulcer of the intestine, with smooth edges, was found, and the coroner gave a verdict of traumatic ulceration of the bowel, with which he, however, could scarcely agree. It was possible that the smooth edges resulted from postmortem digestion.

Dr. Roberts related the case of a sailor who was struck in the abdomen by a pulley-block, and later died of a small, smooth-edged ulcer of the intestine. He thought there might have been a contusion of the bowel with subsequent ulceration.

Dr. Cattell thought it was possible to have perforation of the intestine from poison, *e. g.*, from arsenic, and he had just observed a case of this kind in a suicide from arsenic, in which an ulcer existed at the ileocecal valve. Dr. Lainé and he had seen a case of such a nature several years ago that had led to much discussion. *November* 12, 1896.

28. *Tuberculous ulcers of intestine with a vermiform appendix three eighths of an inch long.*

Exhibited by W. S. Newcomet, M.D.

Tuberculous ulcers of the intestines are not rare, as about one-third of all phthisis-cases present a few, which are usually in the ileum or cecum, although they may be scattered along the whole intestinal tract, as in this case. The mesenteric glands are stony-hard in this instance; the vermiform appendix is only about three-eighths of an inch in length. A gallstone, weighing 15 grams, was also found. The history is as follows:

D. E. M., aged thirty-five years; born in Philadelphia; carpenter; married. Father died of asthma; mother living; three sisters and three brothers living and well; one sister died of phthisis shortly after confinement.

Personal history: The man had measles, whooping-cough, diphtheria, and several attacks of malaria.

Present illness: Late in the summer of 1895, while captain on one of the river boats and exposed to considerable dampness, he contracted a severe cold. This lasted through the winter; the cough was severe; he had frequent chills and sweats, at the same time profuse diarrhea. Toward spring he improved, but the diarrhea and pain in his stomach continued, although not quite so bad as before. He had lost in that time about twenty pounds. He had a short hacking cough, most frequent at night. Very little expectoration.

Physical signs: He was very much emaciated. There was more or less consolidation scattered over both lungs. The stools numbered five or six a day, sometimes more, were very loose, although there were hard lumps in the soft mass, which gave him great pain, and some blood would be passed now and then. He appeared to improve for a time, the stools being reduced in number to one or two a day, but they remained soft; this continued for some time; then he went out to the country. Here again he was taken with diarrhea; he brought me some of the lumps from his stools, expressing the opinion that he thought they were worms. They were round, about the size of a hickory-nut, had a yellowish color; when opened they were perfectly white; examination by the microscope proved the material was milk.

The lungs began to soften, and he spat up a very fetid, rather thin,

mucus; he went on from bad to worse, and died of exhaustion. The postmortem was made about fourteen hours after death.

The *lungs* were both adherent to the pleura, leaving no trace of the pleural cavity. They were completely studded with tubercles. A large cavity had developed at the right apex, with several smaller ones in the lower lobes. The left apex contained a cavity, but not so large as the right.

Intestines presented ulcers through their entire length, varying in size, some as large as a quarter of a dollar. The intestines were bound together in some spots by adhesions, especially around the cecum and ileum. These bands were very firm and evidently old. The appendix appeared to be absent, but upon carefully examining the cecum it was found, but so very small that its relation had to be studied before it could be stated to be the appendix.

The mesenteric glands were enlarged, and some were very hard; when broken open the interior was filled with a soft cheesy mass.

The *liver* contained a few tubercles. The gall-bladder was not yellowish as it is usually, but was white. It was collapsed in its anterior portion, while the portion toward the duct was distended, and was much thinner than the collapsed portion. Upon opening it, one large stone and eleven smaller ones and some thin white mucus were found. The larger stone seemed to be a mass of smaller ones bound together by a crystalline mass, probably cholesterin. It weighed 15 grams. The *kidneys* contained a few tubercles, and were softer than usual; the capsule stripped easily. The pericardium over the heart was thickened in patches, as if he had had an old pericarditis. The heart-valves were compressed; there were some atheromatous patches in the aorta and upon the mitral and aortic valves. *November* 12, 1896.

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29. *Primary carcinoma of the esophagus, perforation of right bronchus, extension to the lung.*

Exhibited by J. P. Arnold, M.D.

The specimen presented was obtained from a man I have had under observation for the past eight months.

The patient was a male, aged sixty-two years; admitted to the Presbyterian Hospital, August 20, 1895, complaining of rapid loss of flesh for past four months, constant vomiting, pain behind the middle of the

sternum, and pain in the epigastrium. He has, in the past four months, scarcely been able to swallow any solid food, and has lived on bread and milk. He has a sensation after attempting to take solid food as if the food lodged at a point which he localizes behind the lower third of the sternum. The family history is negative, except that one brother died of cancer of the stomach. A point of interest in the patient's own history is that he was a sailor and was accustomed to the use of large quantities of strong liquor, especially rum. The mode of onset was described as follows: About a year or a year and a half ago he noticed that immediately after eating he would have a feeling of intense smarting behind the middle of the sternum. In a short time food apparently lodged at this point and would be regurgitated. The man, upon admission, was much emaciated and very weak. His frame was that of a man who should weigh 160 pounds. His weight at that time was only 115 pounds. Blood-count showed a little over 3,000,000 red cells. The esophageal tube stopped twelve inches from the teeth. The vomitus, probably only from esophagus, gave no reaction for free mineral acid. Lactic acid was present.

He remained in the ward until December 24, 1895. During this time he gained fourteen pounds in weight, and improved considerably in general condition. On November 19th a bougie the size of a small acorn could be passed into the stomach.

On February 15th of present year patient was readmitted much emaciated; weight 110 pounds. He had scarcely been able to swallow anything for three weeks. An esophageal bougie of medium size could be passed into the stomach, though considerable resistance was met with at level of cricoid cartilage and just before the cardiac orifice was reached. He began, on March 2d, to cough and complain of pain over right side. There was some dulness over the lower lobe of right lung. Expectoration became quite profuse and very offensive. He progressively failed and died of exhaustion May 16, 1896.

Autopsy. The points of interest were as follows: The tissues of the mediastinum were adherent in one mass, and tightly attached to the right lung. The lower lobe of the right lung was so tightly adherent, both to the thoracic and diaphragmatic pleura, that it could only be removed by tearing its substance. The whole was of a greenish color and was consolidated. The mediastinal glands were much enlarged. The mediastinal tissues, with the aorta, trachea, esophagus, and as much as possible of the lower lobe of the right lung, were removed *en masse*. The stomach was very small; capacity not more than one and a half pints.

Upon opening the esophagus a new growth, encircling the tube like a broad belt, was seen beginning 6 cm. above the cardia and extending upward a distance of 10 cm. It completely encircled the tube, and the greater part of its surface was ulcerated. The obstruction was most marked at the lower end of the growth. The upper part of the growth was not ulcerated. It was hard, whitish, and presented papillary elevations. The esophageal wall was perforated by the ulcerative process, and the opening communicated with the right bronchus. Sections of the growth studied microscopically showed it to be a squamous epithelioma. The right pneumogastric nerve was involved in the growth near the site of perforation. This probably accounted for the slow pulse observed during March and April. The pulse varied from 50 to 60, though the temperature was between 100° and 102° F.

The liver was fatty, but presented no nodules. The mesenteric glands were enlarged but not hard. The kidneys showed a parenchymatous nephritis and a catarrhal pyelitis. There were a few small nodules in the left kidney and upon the peritoneum of the mesentery.

Cancer, though it is the most frequent disease of the esophagus, is not of very common occurrence, and is most frequently primary. Zenker and Von Ziemssen[1] in 5079 autopsies found it present in 0.36 per cent. The growth was primary in 0.25 per cent. The character of the growth appears always to be that of a flat-celled epithelioma, though E. Rindfleisch reports a softer and more superficial form.

The most frequent site, according to Gross,[2] is behind the larynx. Osler[3] gives the upper third as the most frequent situation. According to Zenker and Von Ziemssen and Pepper[4] the growth is usually found in the lower third, the place of election, according to Pepper, being, in the order of frequency, just above the cardia, at the crossing of the left bronchus, and the region of the cricoid cartilage. It is not unusual for more than four inches of the tube to be involved. Ulceration usually occurs, and begins early. In the later stages signs of obstruction may decrease, due to the progress of the ulcerative process. This occurred in the present case. When first admitted, not even the smallest bougie could be passed. Later, a medium-sized bougie passed without difficulty into the stomach. Extension may occur to stomach, pharynx, lungs, trachea, lymph-glands, heart, aorta, or the vertebral column may be involved, causing paraplegia, as in a case reported by Ross.[5] Perforation into the subclavican[6] with fatal hemorrhage, or into an intercostal[7] artery may occur. A case is reported by Homans,[8] which opened externally and into the trachea. Metastasis, according to Pepper,[4] is less common than in other cases of carcinoma. According to Strümpell[9]

metastatic growths in the liver, lungs, kidney, pancreas, bones, and brain are not uncommon. In this case they occurred in one kidney and on the peritoneum. The predisposing causes given by Strümpell[9] are alcoholism, swallowing foreign bodies, scars of old ulcers, and heredity. In the present case there was a history of alcoholism, and one brother died of cancer of the stomach.

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[5] Canadian Medical and Surgical Journal, 1877–'78, No. vi. p. 402. G. Ross.
[6] Transactions London Pathological Society, 1860–'66, vol. xii. p. 108. Dickinson.
[7] Transactions London Pathological Society, 1857–'58, vol. ix. p. 202. S. Jones.
[8] Boston Medical and Surgical Journal, 1866, No. lxxiv. p. 502. C. Homans.
[9] Text-book of Medicine, Strümpell, second edition, p. 346.

December 10, 1896.

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30. *Abscess of the liver.*

Exhibited by S. Solis–Cohen, M.D.

The patient, an Englishman, a tinsmith by occupation, aged fifty-two years, was admitted to the Philadelphia Hospital a few weeks ago. Three weeks before admission he had dysentery. On entrance he complained of pain in the epigastrium and in the hepatic region, which pain had existed four days. There were redness and edema in the epigastric region, the latter especially to the left of the median line; there was also marked tenderness over the liver. Just to the left of the median line a mass the size of an orange was noted; it was dull on percussion and tender. The upper boundary shaded into the liver dulness. The temperature was hectic in type, associated with sweats and chills. The bowels were loose, and once the patient passed a half-pint of blood. At the operation, performed by Drs. Hearn and Da Costa, on October 25th, an abscess of the left lobe of the liver and a general peritonitis were found. The patient died on the following morning.

Autopsy. A recent general peritonitis was found, with rust-colored pus in the dependent parts of the abdomen. The intestines were agglutinated by layers of lymph. The cavity in the liver consisted of two branches: one extending upward and backward and the other to the right. In this region the capsule of the liver was adherent to the diaphragm and abdominal wall. Over the right lobe there were firm ad-

hesions between the diaphragm and the liver. A large abscess occupied the anterior portion of the right lobe. The gall-bladder was adherent by old bands of adhesions to arch of duodenum and omentum. It extended about one inch below the liver margin. The entire surface showed recent peritonitis; upper surface was adherent to the diaphragm. There was an abscess in the anterior edge of left lobe, which had been opened and drained by the operation. Three abscesses were found in the right lobe, separate and distinct, one being at the right posterior limit of the right lobe; another was in the central section one inch from the margin, and a third one occupied the entire upper anterior portion. These abscesses varied in size from one to three inches. Throughout the liver-structure there were several areas of necrosis, the beginning of additional abscesses. Weight of liver 2370 grams.

Small intestines showed no ulcerations, but distinct hyperemia. Large intestine, including valve, showed extensive ulceration.

Right pleural cavity obliterated posteriorly and over entire base. On removing lung a pus cavity was opened, part of which was in the lower lobe and the rest in the upper part of the right lobe of the liver. The pulmonary portion was very irregular in outline, being composed of many small pockets. *December* 10, 1896.

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31. *Perforation of a tuberculous ulcer of the intestine.*

Exhibited by S. Solis-Cohen, M.D.

One can hardly conceive of a more widespread plastic and purulent peritonitis than was found in this case. The omentum was drawn up and retracted, and the amount of lymph had so increased the bulk of this portion of the abdominal cavity that it looked somewhat like a peritoneal endothelioma. On lifting up this mass fibropurulent pockets, with shred-like adhesions, were found over the spleen, liver, stomach, etc. The point of perforation in the small intestine was found in an ulcer about five feet above the ileocecal valve. Tubercle-bacilli had been found in the sputum, and a typical caseous pneumonia, with small cavity-formation, and small cheesy tubercles were found in various portions of the lungs. The kidneys showed a parenchymatous nephritis, and the heart was below normal in size. *December* 10, 1896.

32. *Tuberculosis of the esophagus, with cancerous infiltration.*

Exhibited by D. L. Edsall, M.D.

L. S., aged forty-two years; born in Russia and came to America in 1888; occupation, cigarmaker.

In March, 1895, he caught a severe cold, which did not persist as such, but soon after he noticed a feeling of constriction and oppression in his throat, with no other symptoms. Later he found swallowing became difficult, and increasingly so, and one day in the spring a piece of meat caught in his throat, and upon attempting to force it on with his finger he vomited, the vomit containing blood. With this exception he had no vomiting at any time. The dysphagia increased until, in July, he could not swallow milk, but this improved later, though he could never subsequently swallow solid food. In July his voice became weak and hoarse, and this increased continuously. Severe pain had never been present, and he had worked as usual up to July.

In September he was admitted to the Jefferson Hospital, when a gastric fistula was established. He recovered well from the operation, and left there six weeks after admission. He gave no history of any injury to the esophagus or of syphilis.

When admitted to the University Hospital he was much emaciated, anemic, his voice very weak and hoarse, his pulse weak, and temperature subnormal. He complained only of his weakness and absolute inability to swallow, and of a feeling of oppression in the chest that he could not localize. He had, however, a frequent hawking cough with profuse sputum that was largely saliva with some mucus and pus. The examination of the esophagus by bougie could not at first be carried out, owing to his great excitability; but subsequent examination showed that there was an impassable stricture at the beginning of the esophagus. The cervical glands were enlarged. The percussion note over both lungs anteriorly was hyperresonant. Posteriorly there was slight dulness at the vertebral border of the left scapula and beneath the ridge of its spine; elsewhere the note was normal. The tactile fremitus was alike on the two sides. Air seemed to enter both lungs equally. The breath-sounds were themselves normal, but accompanied by some dry râles and inspiratory crepitations in both apices posteriorly. The heart area was normal; the sounds clear and rapid; the second sound somewhat accentuated.

The stomach-area, determined by percussion and auscultatory percus-

sion, was somewhat enlarged, reaching from the fifth interspace downward to a finger's breadth above the umbilicus. There was a gastric fistula, and he was being fed through a tube.

Examination of his sputum for tubercle-bacilli was negative. His urine was normal in appearance and contained neither albumin nor sugar. The examination of his blood showed 3,282,000 red corpuscles, 6250 white, and 40 per cent. hemoglobin.

Dr. Grayson reported after laryngoscopic examination: Thickening of the ary-epiglottic folds and the vocal cords, and a complete occlusion of the esophagus at its commencement, with no ulcerating or proliferating tissue. The condition was held to be tuberculous, and operation was considered with a view of removing any glands that might be causing the obstruction, and of reopening the lumen.

He improved for a few days upon tonics and free liquid diet. His cough increased, however, and his temperature became a little irregular and his respirations more frequent. Ten days after admission tubercle-bacilli were found in the sputum, and he began to lose a little flesh, and was perceptibly growing weaker. His breathing was so shallow and his voice so weak that it was difficult to determine an increase in the lung-signs. But about six weeks after admission he showed slight dulness with prolonged expiration posteriorly at the top of the right lower lobe. At this time abscesses formed in two of the enlarged glands on the right side of the neck, and were opened; the pus showed no tubercle-bacilli but many staphylococci.

The lung-signs increased continuously, though slightly, and there was constant loss of flesh and strength until, on March 18th, he grew decidedly worse, with signs of catarrhal pneumonia of the right side. Next day there was outspoken pneumonia of the right base, and the left base partially consolidated, and on the 21st he died of failing respiration and heart.

The repeated examinations of his stomach-contents gave interesting results. Various test-meals, as milk, milk and eggs, Boas' oatmeal soup, and the latter with egg-albumin added, and the Ewald-Boas bread and water, were used. The results varied but slightly at any time, usually showing an entire absence of free hydrochloric acid with a small amount of combined chlorids. Once there was a slight trace of free hydrochloric acid, tested with phloroglucin-vanillin, while the total acidity, expressed in terms of its equivalent in one-tenth normal NaOH, after the manner of Ewald, never reached more than 10. Quantitative estimations of the free and combined HCl, using the method attributed by v. Leube to Braun and v. Jaksch's modification of Sjoquirst's method,

gave, at various times, from 0.0329 per cent. to 0.01582 per cent. total HCl. Tests were made at intervals of fifteen, thirty, forty-five, sixty, seventy-five, and ninety minutes when practicable, and the amount of HCl was greatest after about one hour. Usually nothing was recovered after longer than one hour, and frequently after more than half an hour the whole quantity administered had disappeared. This was explained postmortem by the hour-glass shape of the stomach; probably a considerable quantity often remained, but could not be reached by the tube.

Starches never showed transformation into sugars. The gastric contents never showed any active digestion of albumin, and tests for peptone at any interval were always negative. The milk-curdling ferment was present, as was its zymogen, but both in small quantity. With Boas' method for their quantitative determination, a dilution of one-fifth was sufficient to stop the action of the ferment, and one-fifteenth of its zymogen.

Lactic acid was never present, either by Uffelmann's or Boas' tests, probably because the contents were too short a time in the stomach to allow of its formation. Ewald has examined the HCl production in a number of cases of cancer of the cardia upon whom gastric fistula had been made, and found it always absent. Likewise in a number of cases examined by means of the stomach-tube, he found no digestive action.

Neschaieff had like results in four cases of esophageal cancer. Hence Ewald considers that the introduction of food into the stomach through the gastric fistula would lead only to its fermentation, and advises that the fistula be made as near as possible to the pylorus, so that the tube, and thus the food, can be carried through the pylorus directly into the duodenum. On the contrary, Riegel has examined two cases, Sidney Martin one, and Boas one (the location of the disease not mentioned), Boas one of the cardia, and Mintz three of the cardia, in all of which the HCl was practically normal in amount. In one case of Mintz's the motor activity of the stomach was below normal, and in another there was no free HCl, but a normal quantity of combined chlorids, while Riegel had one case in which the secretion was at one time lessened but afterward normal.

There seems, therefore, no definite present knowledge of an ill effect of even cancer of the cardia upon the gastric functions, while it would appear very unlikely that a cancerous infiltration of a stenosis, in the location found postmortem in the case under consideration, should have any specifically harmful action. The conditions of the gastric functions were more probably due to the man's general condition and to the absolute nature of the stenosis, which not only prevented his taking nourish-

ment in the normal manner and condition, but, perhaps quite as important, prevented the entrance of the saliva into his stomach with or without his food, as well as its mastication. Sticker, Biernacki, and Friedenwald have shown that test-meals administered through a tube and no saliva swallowed showed much less activity of the stomach-functions than when the same meal was masticated and insalivated.

The postmortem findings were as follows: Body much emaciated. Fistulous opening in the epigastrium. The deep group of cervical glands cheesy and suppurating.

Thorax. About 200 c.c. clear serum in the right pleural cavity; a small amount in the left.

Pericardium moderately distended with straw-colored fluid.

Heart. Weight, 160 grams; small and dark; visceral pericardium wrinkled. Subpericardial fat along bloodvessels white and gelatinous; valves healthy.

Lungs. Right apex firmly adherent to the deep cervical tissues. On section it contained a number of hard sclerotic and calcareous nodules. The lower part of the upper lobe normal. The middle lobe intensely edematous. The lower lobe congested, edematous, and irregularly consolidated in lobular areas, at its apex some thickening. The left apex less adherent, in other respects similar to the right. Bullous emphysema of the superficial portions, distinct lung-stones in places. Lower lobe similar to the lower lobe of the right lung, at its topmost part consolidation of fibrous character, and on section tubercle-like areas.

Abdomen. Firm adhesions between the stomach and abdominal wall for a half-inch about the site of the fistula, otherwise the peritoneum normal. Kidneys, suprarenals, and spleen normal. Liver small and congested.

The esophagus, larynx, and stomach removed *en masse.* The stomach hour glass-shaped, due apparently to contraction of the new-formed tissue at the seat of operation. The pyloric portion the size of a small orange. The part above the constriction—the body and fundus—was alone about the size of a normal organ; contents were gas and curds of milk.

The *esophagus* patulous and normal as high up as the level of the arch of the aorta. Above this and up to the junction with the pharynx the lumen had entirely disappeared, and its situation could not be traced. The surrounding tissues and esophageal walls had formed a band of firm fibrous tissue about a half-inch in thickness and one and a half-inches in breadth. The whole mass was firmly adherent to the vertebra.

The *larynx*. The ary-epiglottic folds were thickened and edematous. Cricoid cartilage appeared thickened.

Pathologic diagnosis: Tuberculosis of the cervical glands, larynx, esophagus (?), and lungs. Deglutition-pneumonia.

The histologic examination of the lungs quite confirmed the macroscopic diagnosis of old and recent tuberculosis with catarrhal pneumonia at the bases.

The sections of the growth from portions taken from the junction with the healthy mucous membrane show at once cancerous tissue in a general basis of fibrous tissue. This fibrous tissue is exceedingly dense, arranged in bands with a little areolar tissue between the layers, and in this areolar tissue and about the cancer-nests there is a marked, often quite dense, round-celled infiltration, here and there somewhat in clumps, but not characteristic. The epithelial cells are, at the periphery, almost entirely in nests, in some places irregularly scattered as if remnants of the original mucous membrane. Below the free edge there is not so distinct an alveolar arrangement, but there are long tongues of cancerous tissue extending into the here still more compact fibrous tissue. There is an extremely large number of pearly bodies in and near the cancer-nests and prolongations, and in some of them a good deal of formless débris or a goodly number of round cells among the epithelial. In numerous places there are well-defined clusters of round cells with sometimes a few rather irregularly arranged epithelial cells in or near the center. Sometimes a distinctly formed nest is seen at this point. Sections from deeper within the general mass show, at the end toward the free edge, the process in much the same condition as in the deeper portions of those described. The round cell infiltration is, however, less dense, and the fibrous tissue more irregularly arranged. This arrangement ceases rather sharply and the fibrous tissue beyond is arranged transversely and in distinct bands, separated by aveolar tissue, all freely infiltrated with round cells, but showing almost no cancerous tissue. In some sections there are no epithelial cells separate or in groups, in some one or two nests, and in a few three or four, but never a large number. But the round cells here and, more especially, on the lateral borders, assume a more interesting character. They are frequently collected in small, dense masses, sometimes not well outlined, but occasionally very well defined and irregularly round, with an occasional giant-cell near or beyond the periphery of the group. Some of these tubercle-like masses are undergoing fibroid changes, many are situated at a distance from the cancerous tissue, so that they do not seem dependent upon its irritant action for their origin, and are much more marked than where cancer-

nests are plentiful. Staining the sections for tubercle-bacilli gave negative results in all but two sections, in which bacilli were very sparsely found. It seems probable from these appearances that the original cause of disease was tuberculosis, and that this had caused the fibrous occlusion, while the cancer was secondary—the history of cancer here and its inexorably continuous progress everywhere being strongly against its having caused this fibroid condition with no ulceration and, in the center at least, cured as to its active effects.

Trauma and syphilis seem excluded. He had no history or clinical or pathologic signs of the latter, and no history of any injury, and injury could scarcely give rise to so extensive a lesion without leaving a memory of it. Whether it was originally a lesion of the wall seems somewhat doubtful, but, from the history and extent, probable. Compression from enlarged glands must also be considered, but this is a much rarer cause of stenosis of the esophagus than is commonly supposed, although Vesalius first suggested it six generations and more ago. Barety found the esophagus but once involved in 26 cases of compression by enlarged bronchial glands. Hofemokl had two cases, and could find but one other of distinct stenosis and that not severe. Kämer increased the figures two years later with two cases of his own (one not examined postmortem) and nine from literature. If to these we add Habershon's—mentioned but not counted—Barety's, and one reported by Janeway, the number known at that time still remains insignificant. Traction-diverticula with narrowing of the lumen from contracting glands are quite another matter and much more frequent.

But three cases resembling this in microscopic findings have been reported—Tissier's, in which cicatricial tissue surrounded and contracted the esophagus and a perforation communicated with suppurating glands and the right bronchus, the upper and lower borders of the stenosis showing slight cancerous infiltration; and those of Cardna and Zenker, in which there were miliary tubercles in cancerous ulceration. In both the latter, however, the cancer was probably primary. Tuberculosis of the esophagus, *per se*, has been reported occasionally since 1716. The cases of Chvostek, Paulicki, Kraus, Spillman, and especially of Breus, Peter, and Frerichs, are very probably true cases, while Eppinger's is almost undoubtedly so. Since then positive cases have been reported in which the histologic and bacteriologic examinations were complete: one by Weichselbaum, two by Beck, three by Mazzotti, two by Orth, one by Flexner, three by Zenker, and one by Fraenkel; and Henyng has published a case resulting in cure, the means of diagnosis of which I could not examine. In Breus', Paulicki's, Spillmann's, and one of

Zenker's only was any distinct stenosis mentioned. Cases of very extreme stenosis are rare from any cause. Shaw reported a complete stenosis from cancer. Schomaker, Veggie, Pollock, Roe, Paucet, and Forster have reported very extreme degrees from various causes, and Forster's second case, in which there was almost total occlusion of the esophagus from the cricoid downward for two inches, the thick fibrous deposit allowing the passage of a fine probe only, has a great resemblance to the subject of this report. There was, however, no mention of the other conditions or of the origin of the fibrous tissue. The only case found that closely resembles my own is that mentioned as probably the earliest case reported as tuberculosis of the esophagus, the nature of which is, of course, very uncertain. There was, in this, a complete occlusion of the esophagus and conversion of it for several inches into a fibrous band, thought to be due to contraction of enlarged glands, including the esophagus, and the subsequent conversion of the whole into a fibrous mass.

That this extreme condition should be so rare is not surprising. Not only are the causes rare, but cases could only live under especially favorable circumstances—as with a gastric fistula, as in this case—for a sufficient time for a stenosis to pass from one of a moderate degree to a condition of utter occlusion. *January* 1, 1897.

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33. *Carcinoma of the liver and spleen, probably secondary to carcinoma of the ovary.*

Exhibited by David Riesman, M.D.

The patient, M. B., a married woman, aged forty-three years, had been admitted to the wards of Dr. B. C. Hirst, at the Philadelphia Hospital, on account of a large tumor on the right side of the abdomen. She had always been healthy; was married twenty years ago, but never had any children. Five weeks ago she began to have pain on urination, and about this time discovered her tumor. She was constipated and had frequent attacks of vomiting. On vaginal examination the uterus was found normal, but the entire pelvic wall around it was occupied by a dense, hard mass.

Dr. Hirst operated and found a large cyst springing from the left ovary, and, on puncture, withdrew over a quart of fluid. The cyst-wall with the oviduct and ovary on that side were removed, and the

abdomen closed. The extirpated ovary appeared to be the seat of a new growth, which was, however, not examined microscopically. The patient sank after the operation, and died two days later.

At the *autopsy* the spleen, which measured 12 by 8 cm., and weighed 140 grams, was studded throughout with whitish growths, varying in size from a minute point to a small pea. The liver weighed 1420 grams; its surface was slightly roughened by the presence of innumerable small neoplastic nodules. On the under surface a few larger ones were found. At the transverse fissure were two enlarged lymph-glands. On section the liver had the appearance of country-sausage from the presence of countless newgrowths, the white color of which contrasted strongly with the reddish hue of the liver-tissue. Of the other organs the stomach alone was involved, and showed a nodule on its posterior surface.

While we have no definite data as to the primary growth, there is every reason for thinking that it was in the removed ovary.

It is interesting to see the almost complete limitation of the metastatic growths to the liver and spleen, the peritoneum being not at all involved. The hepatic disease may have been secondary to that of the spleen.

January 14, 1898.

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34. *Foreign body from the stomach of a boy.*[1]

Exhibited by Frank White, M.D.

In looking up the literature on this subject I find many cases reported by surgeons; many were cases of insanity of the acute forms, others were old melancholias. The cases reported were operated upon, and the mortality was remarkably low, in view of the fact that in most cases the foreign body had remained in the stomach for some time previous to surgical interference. No attempt has been made to report the many cases of swallowing of false teeth, coins, buttons, etc., that are frequently seen in hospitals, and usually need nothing more than a little time and care in diet, the foreign body soon passing away without producing any special discomfort and apparently without inducing any serious injury to the gastro-intestinal tract. We have records of cases occurring among

[1] I am indebted to Dr. Joseph Sailer for the postmortem notes, to Dr. W. G. Spiller for assistance in obtaining the literature on the subject, and to Dr. M. W. Barr for the privilege of reporting the case.

idiotic children who have been known to eat grass, leaves, "shell-barks," etc.

The question of an operation on this boy was not considered, inasmuch as he had general tuberculosis, probably many months before our knowledge of the foreign body in his stomach.

In view of the fact that the brain of this boy is to be further reported upon by Dr. Sailer, I have given only a few points taken from our etiologic blank and a few of the clinical notes connected with the case.

J. L., aged fifteen years. The history of the case is as follows: He was born at full term; labor difficult. There was deficient animation at birth; he was nourished by his mother. He had a great deal of gastro-intestinal trouble; also had convulsions from about the second week until the third month after birth. He did not walk until two and one-half years old, and never talked except during the last year of his life; he could say possibly half a dozen words indistinctly. He was generally inactive, never becoming interested in any games, nor did he take part in any of the exercises in which other children found so much delight. He was at times cross and irritable, easily angered, and when in this condition was usually quite indifferent to all requests and commands. He spent most of his time walking slowly around, picking up any little thing he could, and seemed to be in fear of some one seeing him. Indeed, he has been known to watch certain things for some time, and, when no one was looking, quickly pick up the desired object, then, concealing it in his clothing, go to some other part of his play-room, and, if unnoticed, seem to enjoy playing with this attractive article, whatever it may have been. A preference seems always to have been displayed for such things as threads, strings, etc. Another peculiarity early manifested in this child was his distaste for any line of food except milk and crackers, and one might truthfully say that in later life this distaste had grown into a decided aversion to any and all other forms of food. He was unusually old in his appearance; his expression was rather pathetic; he had insufficiency of both internal recti muscles, also a considerable degree of myopia, but, unfortunately, a careful examination of the eye-ground had never been made. His head was mesocephalic, lobules of ears adhering to his face, and he had a deep, narrow, hard palate, irregular, decayed teeth, and some tonsillar enlargement. His gait was rather unsteady; patella tendon reflex (?) increased; power of coordination very poor. The father and mother of this child are of English parentage. The father is a stonecutter and fireman, and is of scarcely average intelligence; but the mother, although rather nervous, is quite bright, and does not seem to be in any way mentally

defective. The mother's grandfather became epileptic and insane rather late in life; he had two sisters who died insane. The mother's father became an epileptic when middle-aged, and he had one sister who became insane before middle life. The mother had three sisters, two of whom became insane and died before the age of thirty. A peculiar analogous condition has existed in the different generations ending, as is not unusual, in idiocy. No brothers, so far as we have been able to ascertain, have developed any form of insanity. The great-grandfather and great grandmother were cousins.

J. L. was admitted to the Pennsylvania Training School for Feeble-Minded Children, October 3, 1886. His mother stated that he had always been rather delicate. He was indolent, untidy in his habits, and

had a great desire to eat sticks and strings. He had always been peculiar about his diet, and objected to almost everything except liquids and semisolids. He had measles when five years of age; since that time he had required no treatment except for a few minor troubles.

My attention was called to him on October 2, 1896, for a gradual emaciation and diarrhea. Under treatment the diarrhea was relieved for a few days, only to start again with increased severity.

October 10th. The movements at this time were frequent, watery in consistence, and followed by considerable pain. The case was looked upon as one of general tuberculosis, with probably tuberculous enteritis. On his admission to the hospital, October 12th, he was much emaciated; his temperature ranged from 101° to 103° F. The heart was rapid;

the pulse at the wrist was very feeble. His eyes were congested; vessels extremely tortuous; some photophobia, also convergent strabismus of both eyes.

19*th*. The temperature was normal. The heart was extremely slow and irregular, pulse ranging between 55 and 68.

20*th*. Temperature subnormal. Respirations were slow, regular, and moderately full. Pulse less irregular.

21*st*. Temperature normal. Eyes were less congested. Feet were swollen, showing venous stasis. Twitching of the eyelids; rolling of the head; strabismus absent.

22*d*. The vessels of the eyes were much smaller and less tortuous. The general lethargic condition passed into one of coma, which ended in death.

Autopsy, December 15, 1896, 2.30 P.M. Male, white, aged fifteen years; died 6 A.M., December 14, 1896.

Body greatly emaciated, feet and legs edematous, abdominal veins distended, gluteal regions bright red; anus directed backward, as a result of the formation of a right angle by the sacrum with the axis of the spinal column. Testicles still in inguinal canal. Postmortem discoloration of abdomen; postmortem lividity. Spinal canal contains edematous cellular tissue and considerable fat; the cord apparently normal, the lower part very firm in structure; calvarium thin and dura mater thick. Over both temporal regions the pia is very edematous and the vessels are injected. Beneath each temporal region, apparently in the arachnoidal space, is a large multilocular cyst filled with a clear fluid. The pia which forms the floor is thickened and covered with trabeculæ, and the convolutions of the temporosphenoidal lobes are compressed and the sulci shallower. The cerebral arteries and all the vessels of the base lying anteriorly to these cysts are intensely injected, and the vessels lying posteriorly are depleted. The abdominal organs are normal in arrangement. Diaphragm, right, at fourth rib; left, at third interspace. The sternum is adherent to the right pleura by granular and purulent adhesions; the left pleural cavity contains no adhesions, but the visceral layer is covered by fine miliary tubercles. The superior mediastinum is filled with greatly enlarged glands that surround the vessels and the trachea, slightly compressing the latter. The pericardial fluid is normal in amount and the epicardium normal. The foramen ovale is not patulous; the tricuspid valve admits two fingers and the mitral valve one finger. The endocardium of all cavities is normal; the myocardium contains a few cicatrices. Upon the epicardium over the left ventricle anteriorly, near the base, there is a single white nodule

dipping into the myocardium; the left lung contains numerous tuberculous nodules of various sizes, the majority being miliary, a few large and undergoing cheesy degeneration. The bronchial glands are enlarged and cheesy; the right lung is surrounded by a greatly thickened pleura that is undergoing cheesy degeneration without cavity-formation, and contains enormous numbers of miliary tubercles. The upper and lower surfaces of the right half of the diaphragm are covered with large cheesy nodules extending deeply into the substance of the muscle. The spleen is small and firm; capsule pale, and in parts adherent to the abdominal wall; there are three deep notches on the lower edge. The left kidney is of normal size; capsule strips readily; relation between cortex and medulla normal. Cortex pale; pyramids distinct; a recent anemic infarct in cortex; a small white nodule in the substance. The pelvis is normal. The right kidney is of the same general condition, but there are neither tubercles nor infarcts. The suprarenal bodies show considerable fatty degeneration of cortex. The pancreas is very dense and apparently sclerotic. The stomach is enlarged and filled with a solid mass, which, upon being opened, proves to consist of wood, straw, grass, and string; its weight is six and a half ounces; its length, five and one-half inches; thickness, two and one-half inches; it is firmly agglutinated and moulded to the form of the stomach; toward the cardiac end the wall of the stomach is greatly thickened and slightly dilated. The small intestines show two white nodules; they contain feces, and the ileum is intensely congested. The mucous membrane of the large intestines in a few areas is thickened and slightly eroded. There are neither tubercles nor ulcers. The lymphatic glands of the mesocolon are greatly enlarged, and the corresponding part of the colon is injected. The liver is pale and contains a few hemorrhagic infarcts. In the capsule are some miliary tubercles. The ascending cava just above the junction with the hepatic vein contains a thrombus that is adherent to the wall in both directions, but no adherent secondary thrombi are found in the vein. The thrombus contains no areas of softening. The bladder-walls are thickened and slightly rugous.

Pathologic diagnosis. Tuberculosis of the lungs; general miliary tuberculosis; cheesy tuberculosis of right lung, right pleura, and right diaphragm; tuberculosis and caseation of superior mediastinal glands; obstruction of gastric lumen by slowly forming foreign body, with hypertrophy of wall at cardiac end; renal anemia; renal and hepatic infarcts; tuberculous mesenteric glands; thrombosis of the ascending cava *ex cachexia.*

Literature.

1. Med. Exam. Cat. of the Surgeon-General's Office, 1884. Neal operated on a patient, removing a large piece of lead, curing the case.

2. Berliner klin. Wochenschrift, 1880, p. 539. Belle reports the case of a man, aged fifty-eight, who had a self-made cleansing apparatus for the pharynx, which in some manner became broken, was swallowed, and remained in the stomach sixteen hours. First an esophagotomy was done, then a gastrotomy; death followed in five days. Length of wire 31½ centimeters.

3. Medical Press and Circular, October 12, 1881; Virchow's Jahresbericht, 1881, ii. p. 40. Fleury reports the case of a man, aged forty-two, who while playing with a fork swallowed it. The prongs stuck into the muscular wall of the stomach; the handle also was impacted. The fork was in the stomach two months; operation was followed by recovery.

4. Bulletin de l'Académie de Médecine, August 24, 1886. Solallion reports a case: A man, aged twenty-five, swallowed a fork 21 centimeters long and weighing 59 grammes. Fifteen days afterward a positive diagnosis was made with an electromagnet; operation; no change in stomach-walls; cured.

5. Lancet, 1891, vol i. p. 249; British Medical Journal, 1893. Report of a case by Lansen, of an insane man, aged thirty-seven, who tried to commit suicide by swallowing a wire 7½ inches long, also the handle from a clay pipe 2½ inches long. Length of time foreign body was in stomach is not given; he was operated upon and cured.

6. Boston Medical and Surgical Journal, 1886, vol. ii. p 569. Richardson reports a man as having swallowed (during sleep) four front teeth from the left upper jaw. These teeth remained in the stomach eleven months; at the time of operation no changes were found in the wall of the stomach; cured.

7. St. Louis Medical and Surgical Journal, 1887; Berliner klin. Wochenschrift. Beranays reports a German tailor and sword swallower, aged twenty-eight, who swallowed a knife 24 centimeters long; it remained in the stomach a few hours; operation; cured.

8. Langenbeck's Archiv, 1887, 37, p. 283. Stetzner reports a man, aged twenty-five, who was arrested for attempting to commit suicide; six pieces of wood from 8 to 10 centimeters long and from ¾ to 1½ centimeters thick remained in the stomach twenty-three days.

9. New York Medical Journal, 1887, vol. ii. p. 481. Bull reports the case of a boy, aged sixteen; a peach-stone was swallowed and lodged in the esophagus under the heart. Nine days afterward an opening was made in the stomach, an instrument containing a sponge was introduced into the esophagus, and the foreign body was removed through the mouth.

10. Nord. med. Arkives, 1888; Schmidt's Jahrbücher, 218. Berg reports a case having swallowed a bunch of hair weighing 900 grammes; time not given; operation; cured.

11. Journal American Medical Association, 1888; Centralblatt f. Chirurgie, No. 48. Witte reports a woman, aged thirty-one, who during an attack of acute mania swallowed a four-tined fork, seven and a half inches long, which remained in the stomach three months. Perforation of the anterior wall of the stomach; operation; cured.

12. Bulletin de Chirurgie, 1889, p. 436; Virchow's Jahresbericht, 2, p. 524. Ferrier reports a butcher boy, aged nineteen, who swallowed a fork 22 centimeters long, while trying to push a piece of orange down the pharynx; operation twenty-four hours afterward; cured.

13. Langenbeck's Archiv, 1889, vol. xxxvii. p. 169. Hashimoto reports a hair-dresser, aged thirty-six, who swallowed a Japanese tooth-brush, which was about the thickness of a lead-pencil. The brush remained in the stomach ten days; the anterior wall was perforated; operation; death on the third day from peritonitis.

14. Skearek. Inaugural Dissertation, Bonn, 1889. Trendelenburg reports a case of a man having swallowed a set of false teeth, which remained in the stomach a few hours; operation; cured.

15. Periar. Virchow's Jahresbericht, 2, p. 514. A German, aged thirty-two, swallowed a German spoon, which remained in the stomach twenty-four days. Diagnosis made with an electromagnet; operation; cured.

16. Boston Medical and Surgical Journal, 1890, 123, 8, 177. Virchow's Jahresb ericht. 2, p. 514. Richardson reports a woman, aged thirty-one, having swallowed an open safety-pin; it lodged in posterior wall of stomach for four days; operation; cured.

17. Semaine Médicale, 1891; Schmidt's Jahrbücher, 232, p. 174. Nancy and Heidenrich report a man, aged twenty-nine, a sword swallower, who swallowed a silver spoon 20 centimeters long, weight 27 grammes. It was in the stomach fourteen days; operation; cured.

18. Finney. Johns Hopkins Hospital Bulletin, October 3, 1892; Virchow's Jahresbericht, p. 1447. A man, aged forty-nine, swallowed a peach-stone, which lodged in the esophagus; it was removed through the stomach. Length of time in esophagus five days; operation; cured.

19. Lancet, 1893, vol. i. p. 20; Centralblatt f. Chirurgie, 1883, No. 36. Cant reports that a woman with suicidal mania swallowed a razor; in stomach six days; operation; death on fifth day from hemorrhage and abscess of the stomach-wall.

20. Lancet, 1894, vol. i. p. 734. Wallace reports that a woman, aged 28, during sleep swallowed a plate of false teeth, which lodged in the lower part of the esophagus. The foreign body was removed by introducing an instrument into the esophagus through the stomach

21. Lancet, 1894, vol. ii. p. 432. Geunnel operated on a patient, aged forty-three, suffering from chronic mania, and removed 192 nails and large bunch of hair, weighing 1 pound 9½ ozs. Front wall of stomach much eroded, but not perforated. The nails were in numerous places impacted in the walls of the stomach, necessitating extensive dissection. Death followed in four hours, from shock.

22. Girl, aged ten years, swallowed forty-two iron nails, ninety-three brass and lead sticks, twelve large nails, three shirt buttons, a safety-pin, and a needle. No changes in stomach-wall; cured.

23. Deutsche med. Wochenschrift, 1894. Czerny reports that a laborer, aged thirty-two, swallowed three closed pocket-knives; in stomach forty hours; no changes in stomach-wall; cured. He afterward swallowed two knives.

24. Lancet, April 6, 1895, p. 904. Swain reports the case of a girl, aged twenty, who swallowed a bunch of hair weighing five pounds and three ounces; no change in stomach-wall; cured.

25. Centralblatt f. Chirurgie, 1895, p. 4. Aufschulen reports that a woman, aged thirty, while weaving cowhair, swallowed a bunch of hair 7.006 centimeters in diameter; weight 120 grammes. No change in stomach-wall; extended growth within the peritoneum; cured.

26. Norton. Annals of Surgery, April, 1896, p. 415; Centralblatt f. Chirurgie, No. 28. Bristol reports the case of a man, aged twenty-seven, who swallowed a plate with two upper front teeth; in the stomach a year and three-quarters; death on the seventh day, of peritonitis

27. Centralblatt f Chirurgie, No. 31. Phelzuem reports the case of a girl aged seventeen, with the habit of biting off her own and other girls' hair-plaits, swallowed a bunch of hair the size of a goose-egg, weighing 180 grammes; no change in stomach-walls; cured.

Discussion.—Dr. Eshner thought the case extremely interesting, especially in view of the numerous instances reported in hysteric women in whom balls of hair had been found in the stomach. By some these had been considered dermoids, but the present case showed that they may come from without.

Dr. Sailer had found the mucous membrane of the stomach on microscopic examination in surprisingly good condition, except for a slight excess of muscular tissue, especially at the pylorus, and a few collections of round cells in the muscular layer.

Dr. Stengel spoke of cases of dilatation of the stomach from foreign bodies, in one instance a peach-stone was impacted in the pylorus. An interesting case had been reported recently: A furniture-worker from holding the brushes in his mouth had gradually swallowed a quantity of resin, which collected in the stomach and caused dilatation. In a case reported here some time ago, a palpable tumor in the wall of the stomach had acted as a ball-valve and caused obstruction.

Dr. Coplin was impressed with the regularity with which the fibrils of the body were arranged. *January* 28, 1897.

35. *Hydatid cyst of the liver.*

Exhibited by Frederick A. Packard, M.D.

The specimen shown was found at autopsy upon a patient dead of chronic parenchymatous nephritis. She showed the usual lesions of that condition, and in addition there was found in the liver a rounded tumor, with smooth, bluish surface, projecting beyond the upper surface of the left lobe near to the anterior edge. On examination it was found to be round, of about the size of a marble, 1.5 cm. in diameter, not infiltrating the liver-substance, and composed of a simple cyst-wall and soapy contents. Under the microscope the contents showed great masses of cholesterin-crystals and a very small number of echinococcus-hooklets; no other cysts were present. The patient was a colored woman, aged thirty-four years, a native of and constant resident in the United States. Inasmuch as no suspicion of hydatid disease had been harbored before death, her habits regarding the handling of dogs were not ascertained.

January 28, 1897.

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36. *Syphilis of the liver.*

Exhibited by Alfred Stengel, M.D.

N. C. C., aged sixty-five years, a hotel-keeper, was admitted to the University Hospital, July 18, 1895. He had always been a healthy man, but never worked hard. He used tobacco excessively until two years before, but drank only moderately. He ate very irregularly, and constantly bolted his food without mastication. There was no history of syphilitic infection.

In April, 1894, he began to lose weight, and suffered with a dry cough. The loss of weight went on steadily until it amounted to fifty pounds. He became weak and sallow in color; his appetite varied, sometimes becoming ravenous. His bowels acted irregularly, but he was rarely constipated. Occasionally he vomited, but lately not at all. There was practically no pain.

Physical examination. The patient is emaciated, sallow, and almost cachectic. The abdomen is rather hollowed, and in the position of the lower border of the liver is seen a distinct, nodular tumor which moves with the respirations. Auscultatory percussion determined that the

pylorus lies near the tumor in question or behind it. The liver begins at the fifth interspace and extends 3 cm. below the edge of the ribs. The hepatic dulness is continuous with that of the tumor noted.

The stomach contents were removed after a test-meal, and hydrochloric acid was found present. Subsequently this test was repeated on numerous occasions, and generally there was found a slight amount of hydrochloric acid. Sometimes it was absent. Boas' test-meal was given on three occasions, but lactic acid was always absent. There never was any evidence of retention of food beyond the normal length of time, and the stomach was always empty in the morning before breakfast.

Examination of the blood showed 4,125,000 red corpuscles, 12,500 leukocytes, and 45 per cent. hemoglobin. The red corpuscles were irregular in shape, but there was no marked abnormality.

The urine was normal in color, acid in reaction, and had a specific gravity of 1018. There was no albumin, no sugar, and no casts.

The stomach was distended with air on several occasions, and the lower border was found as low as the umbilicus, but the pylorus was also depressed, so that the descent of the lower border was more likely the result of dislocation than of dilatation. When the stomach was inflated it was easy to determine that the mass on the right side was not connected with the pyloric end, as the latter was separated by a distinct and easily determined interval from the tumor in question.

The patient was placed upon a tonic treatment, and was ordered careful diet and remedies directed to the general disturbances of his stomach, supposed to be the result of chronic gastritis.

There was some improvement for a time, but subsequently the condition relapsed. Finally operation was decided upon, and was performed by Dr. J. William White. The mass was found to be a nodular growth in the anterior surface of the liver and projecting also to a certain extent upon the posterior surface. The stomach was uninvolved. No portions of the growth were removed, but the wound was simply reunited. The patient subsequently improved almost without interruption, and when he finally left the hospital, three months later, had gained over twenty-five pounds in weight, and suffered no gastric or other disturbances.

The patient returned to the hospital the following year, stating that he had improved greatly after leaving the hospital, but had subsequently lost flesh somewhat, and returned on that account. The examination showed nothing additional, though it was very evident that the tumor had decreased in size. The patient was in about the same condition

when he met death through an accident, falling through an elevator shaft to the cellar.

Autopsy disclosed the following conditions among others: The stomach was practically normal, the pylorus entirely free, and the mucosa healthy. The lower surface of the liver presented a partially cicatrized tumor-mass, projecting above the surface as small, rounded, yellowish nodules separated by areas of cicatricial contraction. The upper surface was almost free, although depressions of the surface indicated the position of the tumor visible at the operation. On section into the tumor-mass these were found to be yellowish in color, consistent, and structureless. Considerable fibrous tissue surrounded the nodules, and radiating cicatricial bands were observed.

Microscopically, the tumor itself was found to consist of an irregular connective-tissue stroma with stellate and epithelioid cells. There was no suggestion of carcinoma or of sarcoma in the appearance, and a diagnosis of gumma was, therefore, apparently justified. The other organs presented nothing of interest in the case.

The most important feature in this case was the spontaneous resolution and cicatrization of the tumor. This was continuous and very marked and dated from the time of the operation. Previous to this the mass had steadily increased in size, and the patient's general condition was most unsatisfactory. Immediately after the operation improvement began, and never ceased while the patient was under our observation.

January 28, 1897.

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37. *Amebic abscess of the liver.*

Exhibited by Alfred Stengel, M.D.

The case I have the honor of reporting occurred in the German Hospital, in the service of Dr. Lawrence Wolff. The autopsy was performed by myself.

Male, aged fifty years; single; coal-yard worker; born in Ireland. Father and mother died of unknown causes; one brother killed on railroad; one sister died of dropsy. No history of tuberculosis or cancer in the family.

He had measles, whooping-cough, typhoid fever (walking) when a boy, grippe four or five years ago; not subject to coughs or colds. Denied specific diseases. Used alcohol and tobacco, probably not to excess, but quite regularly—mostly whiskey.

The patient has been sick since July, 1896, with the present trouble; it began with severe diarrhea (ten or twelve stools in twenty-four hours). Now he has about three or four stools in twenty-four hours. At first the stools were very small, slimy, and blood-stained, passed with much straining. Now these conditions are milder, and he seldom, if ever, passes blood; there is no great tenesmus. Vomited a little at first, but not lately. At first he had pain in abdomen, probably at right iliac fossa. Has tenderness over line of colon now. Gurgling in the right iliac fossa. Appetite is fair. Never became jaundiced during this illness. Tongue is slightly coated. Lost much strength and flesh since July. There is a slight mitral systolic murmur transmitted for a short distance to the left. Lungs normal. Urine negative.

Subsequently the following notes were recorded: Never complained of pain over any region of his abdomen. Spleen was not enlarged. No apparent enlargement and no tenderness of the liver. Was not jaundiced. Temperature showed quite decided morning and evening variations, usually of about 2° to 3°; but lately it became subnormal, both morning and evening. Had no chills nor sweats. Stools persistently liquid, greenish or brownish in color, very offensive; from six to eight passages a day, sometimes less. Has had no tenesmus, nor has he passed blood lately. Urine was normal when he was admitted, one month ago, but has not been examined again. Subsequently the patient became very anemic and cachectic, and gradually got weaker and more emaciated. He was in poor condition when admitted and hardly able to walk a few steps.

The patient had a slight nose-bleed during the last weeks of his illness. The last day of life patient had profuse sweating.

Patient was given a test-breakfast (about two weeks before his death), and stomach-contents were found to contain no free HCl, but were acid in reaction.

Autopsy. Six hours after death. Body of an emaciated and sallow white man. The abdomen was somewhat distended. On opening the thoracic cavity a moderate quantity of clear fluid was found in each pleural cavity. The pericardial sac likewise contained a little clear serum. The heart was of average size, but the muscle was soft and flabby. The coronary vessels were very tortuous and projected distinctly. The epicardial fat was in places clear and watery in color (edematous); it was slight in amount. The muscle of the heart was dark-brown in color, and on cross section presented a few areas of fibrous overgrowth (Schwielen), particularly in the papillary muscles. The endocardium was normal. The cavities of the heart were of

average size and contained dark-red clots with some white coagula, extending into the pulmonary artery and aorta. The orifices and valve segments were normal. The lungs were somewhat congested at the bases and rather edematous throughout, but showed no distinct lesions.

The abdomen was distended, the intestines being considerably inflated. The appendix presented a normal appearance. The mesenteric glands were moderately enlarged, and on section were of pinkish color and rather moist. The stomach occupied a normal position, the pylorus being beneath the edge of the liver. On section there was found a little induration of the pyloric ring, but otherwise no abnormality was seen. The small intestines were practically normal, excepting a small portion just above the ileocecal valve where the mucosa was congested. The duodenal papilla was unobstructed. The large intestine was diseased throughout its whole extent. The wall of the gut was uniformly thickened, and on section showed edematous swelling of the mucosa and fibrous hyperplasia of the outer coats. The surface of the mucosa was intensely congested and elevated in a somewhat granular or papillomatous manner. Between the elevations were found large numbers of erosions or ulcerations of irregular outline, most of them somewhat transverse to the length of the gut. The edges were sloping and rather indefinite, and the floor of the ulcers of lighter color than the surrounding (preserved) mucous membrane. They were all quite superficial; and were everywhere so numerous that the picture presented was practically one of alternating elevation with congestion, and erosion, and necrosis of the surface.

In the region of the hepatic flexure, and less so elsewhere, there was in addition some pseudomembranous deposit. This was quite superficial and of yellowish color. The individual patches varied in size from 1 to 3 or 4 cm. in diameter, and were for the most part quite irregular in outline. When the membrane was lifted from its base the underlying mucous membrane was found necrotic. At the hepatic flexure the colon was firmly attached to the under surface of the liver above and to the kidney behind. The adhesion to the liver was absolute, so that the gut was only removable by section; the attachment to the kidney was comparatively slight. In this region the outer coat of the intestine was considerably thickened and fibrous, merging into the wall of fibrous tissue surrounding the abscess of the liver, presently to be described.

The liver was only slightly enlarged, and in its removal an abscess of the right lobe was opened and emptied of its contents. The abscess was found adjacent to the attachment of the hepatic flexure of the colon

and extended upward toward the convexity of the liver. It was spherical in shape, and about 15 to 20 cm. in diameter. The upper wall extended to within 5 or 6 cm. of the upper surface of the right lobe, the lower wall was formed or closed by the attached colon and right kidney, both of these structures being firmly adherent and attached by dense fibrous tissue. At the extreme right the abscess reached the surface of the liver, and in this situation firm fibrous adhesions attached the organ to the abdominal wall. The amount of fibrous tissue was very considerable, the wall of the abscess, including the part attached to the wall of the abdomen, being fully 3 cm. thick. The contents of the abscess were quite offensive in odor, and consisted of yellowish, curdy pus. The inner wall of the abscess was composed of similarly curdy yellow tissue, presenting irregular elevations. After washing out the liquid contents this puriform wall of the abscess had a thickness of from 1 to 2 cm. Outside of this was found a dense fibrous wall of whitish color and of a somewhat striated character. This was from 2 to 3 cm. thick and completely enclosed the abscess. At one point a small focus, containing pus and having a diameter of about one-half cm., was found outside the abscess wall.

The gall-bladder was uninvolved, and the biliary ducts patulous.

The right kidney was uninvolved, though it was intimately attached to the floor of the abscess. Its substance was somewhat pale and the pyramids congested. The opposite organ presented similar appearances.

The right suprarenal gland was uninvolved and of normal appearance. The left was similar. The lymphatic glands in the fissures of the liver and near the head of the pancreas were enlarged, soft, and pinkish in color. The surface of section was moist. The pancreas presented no abnormality. The spleen was somewhat enlarged and soft, and the Malpighian bodies were enlarged and pale. The bladder and prostate gland presented no evidences of disease.

Some of the pus was removed from the abscess-cavity and immediately examined. Various cellular constituents, mostly leukocytes, were discovered, and in addition blood-pigment and much granular detritus, oil-droplets, and other particles of doubtful nature. Of greater interest, however, was the discovery of the ameba coli. This organism was found in moderate abundance, some fields of the microscope (objective $\frac{1}{12}$ N. A. 1.20, eye-piece No. 3, Zeiss) containing two to four amebæ. The examination was conducted upon slightly warmed slides, but without a warming stage. The amebæ were found in motion, though much less active than I have found them in the fresh discharges from the bowel.

The distinction between the granuloplasm and the hyaloplasm was distinctly marked in some of the organisms and not at all in others. Nearly all contained vacuoles, varying in number from one to ten or more. In the latter cases the whole cell seemed converted into a honeycombed body. In the cases in which fewer vacuoles were found these were rather large, but in no case did I find very large vacuoles—that is, such as occupied a considerable part of the cell-body. No contracting or movement of the outline of the vacuoles was detected. The granuloplasm was finely granular and rather homogeneous. Large pigment-masses, such as I have noted in the amebæ of the intestines, were wanting, and no extraneous inclusions were discovered in any of the amebæ seen. The granules showed no movement excepting in mass, when pseudopods were projected. The nucleus of the ameba was visible in but a part of the ones examined. It was best seen in the forms having no vacuoles or but few. The ameboid movements of the living forms were slow but characteristic. The hyaloplasm was first projected in the form of a blunt pseudopod, a gliding or somewhat rolling motion of the protoplasm being observable. The granuloplasm lagged behind, and became almost entirely devoid of its covering of hyaloplasm. When the offshoot had reached about one-quarter or one-fifth the size of the whole body, the granuloplasm suddenly streamed forward into the pseudopod, and the latter at once became filled out and rounded, while the former body of the cell decreased rapidly and remained, for the time, as a hinder extremity, composed of hyaloplasm only.

Sometimes the body became much elongated and quite irregular in outline, and the granular protoplasm seemed thinned out, doubtless on account of flattening of the cell body. It was impossible to determine how long the ameboid movements would have continued under favorable circumstances. The autopsy was made at about 9.45 P.M., and some of the amebæ were still active at 11.45 P.M., the slide having been prepared an hour before that time. Doubtless the movements would have continued much longer with a warm stage.

Besides these actively living forms numerous "encysted" amebæ were found. Upon the whole, however, the latter were in the great minority. Some of the mucopurulent exudate of the intestines, as well as portions of the necrotic membrane, were examined and a few unmistakable amebæ were found. None of these were in motion, and practically all were in the encysted condition.

No measurements of the organisms in the fresh state could be made, but some estimation could be formed from the red blood-corpuscles and pus cells. The amebæ were fairly uniform in size, and at least three or

four times, and perhaps as much as six or seven times, the diameter of a red corpuscle.

Several years since I was able to demonstrate more or less satisfactorily specimens of the ameba coli obtained from cases of dysentery. These amebæ were obtained from the intestinal mucus. I had observed altogether some eight cases of amebic dysentery when I published my paper on this subject in December, 1891, and January, 1892; subsequently (1893) a case of amebic dysentery, with abscess of the liver, came under my observation, but in that instance the amebæ were not satisfactorily demonstrated. The present instance is the first in which I have found the living amebæ in the contents of the hepatic abscess. It may be proper to make a slight *résumé* of the history of this organism and its present position in etiology. Amebæ were first discovered in the stools in a case of enteritis (perhaps dysenteric) by Lambi in 1859. Lewis and Cunningham made further observations in India in 1870, but the etiologic connection of the organism with dysentery was first suggested by a case of Loesch, of St. Petersburg. Subsequently Cunningham, Grassi and Perroncito recorded observations; but the most important studies are those of Kartulis, based upon 500 cases of dysentery in all stages of the disease. After Kartulis numerous studies have been contributed in Europe, Egypt, and America. Of the latter that of Councilman and Lafleur is most important.

It does not seem necessary at the present time to enter upon a minute description of the morphologic and biologic character of this organism. Suffice it to say that the ameba is a protoplasmic body possessing a nucleus and frequently one or more vacuoles. It varies in size from 1 to 30 microns in diameter and shows more or less active ameboid movements. The ameba exists in two states: the active and the encysted. In the latter the body becomes quiescent, and the cell-wall seems tough and frequently presents a double contour. The reproduction of the ameba remains more or less obscure. Resting spores have been discovered in the body of the ameba by Cunningham, and Harris has recently published his observations showing direct division of the ameba.

We are more interested at the present time in the question of the pathogenicity of the organism. Since Kartulis' publications writers have generally admitted without question the specific relation of this organism to dysentery. There is, however, considerable reason to question the soundness of this view, and, to say the least, authors have been disposed to accept the pathogenicity of this organism more easily than is usually done. A number of authors have observed amebæ in conditions other than dysenteric, and, indeed, in healthy persons. Cunning-

ham found the organism in healthy persons as well as in horses and cattle in India, and Grassi found similar amebæ in various diseases as well as in healthy persons, and Blanchard, Calandruccio, Massiutin, Kruse and Pasquale, Quincke and Roos, and especially Schuberg have confirmed these results. It has been suggested by some observers that there are varieties of amebæ, some of them pathogenic, others not so, and Quincke and Roos in particular suggest three varieties: 1, ameba coli, Loesch, the ordinary ameba; 2, ameba coli mitis, the form non-pathogenic for cats and occurring in the mild cases of intestinal inflammation; and, 3, ameba intestini vulgaris, the form found in healthy persons. The distinction of these forms is not based upon accurate morphologic or biologic observations, but rather upon the presence or absence of disease and upon the occurrence or non-occurrence of intestinal irritation in animals when the mucus containing the ameba is injected. It is important to determine, therefore, whether the proposed varieties in reality represent distinct organisms or only forms of the same species having greater or less virulence in consequence of systemic conditions. This question, as well as that of pathogenicity of the ameba itself, must remain a matter of doubt so long as the attempts to obtain the organism in pure culture fail. Cunningham made certain rather crude attempts at cultivation in India, and Kartulis claimed to have obtained pure cultures upon hay-infusion. As far as the latter observations are concerned, other amebæ frequently grow upon this medium, and it is scarcely possible that the cultures obtained after Kartulis' method could have been pure. The injection-experiments to determine the transmission of the disease to animals have, therefore, been carried out with mucus of the intestine containing the ameba, and with the contents of the hepatic abscesses. A number of observers, including Hlava, Loesch, Kartulis, Quincke and Roos, and Kruse and Pasquale, have recorded successful injection-experiments. The cat is the animal most suited to these studies. Other micro-organisms of a vegetable nature are invariably present in the intestinal mucus. Kruse and Pasquale have in particular described three bacilli resembling the typhoid bacillus, bacillus pyocyanus, bacterium clavatum, and a variety of streptococcus. These organisms, however, do not appear to be specific, though they may have pathogenicity. The latter may possibly be brought into prominence after the ameba has exercised its local effects. The injection of the pus from liver-abscesses is of particular interest. These abscesses are frequently sterile as far as the development of cultures prepared in the ordinary manner are concerned. The injection of such sterile pus has been found capable, in some cases, of producing

intestinal lesions, and the conclusion would at first seem to be justified that the ameba alone was active in such cases. This conclusion, however, must be carefully weighed, particularly in the light of observations such as that of Rendu, who described a case in which the necrotic material within the abscess of the liver contained streptococci when studied by the ordinary microscopic methods, but gave no cultures upon artificial media. It is not impossible that such loss of culture-properties may frequently occur, though the organism is still present in the tissue, and may, perhaps, still possess pathogenic power.

The position of the amebæ in the intestinal walls and in the liver and their number strongly favor the suspicion that these organisms are the specific cause of the intestinal disease and of the abscess in the liver. Absolute proof, however, is lacking, and there is no possibility at the present time of carrying out the experimentum crucium.

January 28, 1897.

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38. *Perforation in typhoid fever.*

Exhibited by J. H. Lloyd, M.D., and D. L. Edsall, M.D.

The first case was a girl, aged seventeen years, who was well until three weeks before admission. She had felt weak and forlorn for two weeks, and spent the week previous to admission in bed. Had headache, anorexia, pains in limbs, chilly feelings, and insomnia. Bowels very loose. Rose-spots were found two days before admission. She had no pulmonary symptoms. Upon admission she presented the typhoid facies, tongue coated and dry. A moderate number of spots. Abdomen moderately distended and somewhat tender; spleen enlarged; pulse 120, fairly good. She was slightly delirious at night, but presented no other anxious symptoms until several days after admission, when her lungs became much congested and filled with râles. This cleared up pretty well in a few days, but she began to have marked abdominal pain and tenderness, which continued throughout with varying severity. The plunge baths caused great drops in the temperature, so the temperature of the baths was raised, and their duration shortened to ten minutes, but they still caused excessive reduction. Nothing of moment happened, however, until eleven days after admission, when, during a plunge bath at 8 A.M., she suddenly complained of extreme pain all over the abdomen, and especially in the cecal region. Her pulse went

up ten to twenty beats, and she was quite tender over the abdomen. Her temperature, however, dropped less than after many other baths; her extremities were warm; her abdomen, while a little more distended than usual, was not extremely so, and the liver-dulness was as usual. Her general appearance was not much changed. A consultation was held to consider operation, but the symptoms were considered insufficient to warrant it. Next morning, at another consultation, her condition was about the same. Pain remained a marked symptom, as was tenderness, but these had been already so decided for a week that they were not considered so significant as they might otherwise have been, and operation was again decided against. That evening she grew suddenly worse; her expression became extremely anxious; her extremities cold; her pulse went beyond 160, and her liver-dulness disappeared, while the pain and tenderness decreased, and her temperature pursued its usual irregular course. She died at midnight. Operation, upon its first suggestion to the parents, had been emphatically declined.

The clinical diagnosis was a perforation that had been walled off at first, and had subsequently ruptured a few hours before her death.

Postmortem. The liver-dulness was absent anteriorly but present behind. The abdomen was quite tense, and upon opening it gas escaped freely. The omentum was spread out and lightly adherent and covered with a purulent fluid that filled the pelvis and sides of the abdomen. Handling the intestines caused gas to bubble through the fluid in the right iliac region. The loops of the small intestine in this region were all bound together, in large part, but lightly, though some adhesions were much stronger and apparently several days old. Upon separating these partially a perforation was found almost, or quite, walled in by the adherent loops of intestines, and about eight inches beyond this another perforation lying free to the peritoneal cavity. The edges of the first perforation looked gangrenous. Upon opening the bowels a few small clean ulcers were found in the ascending colon, and a large number in the last four feet of the ileum. Just above the ileocecal valve was one as large as a dollar. The ulcers had nearly completely sloughed, though some still showed shreds not yet separated. Fifty-five cm. from the ileocecal valve was a round ulcer of 2 cm. diameter with a perforation in its center 2 mm. in size; 22 cm. beyond this was another ulcer of the same size and shape, showing a perforation of like position and size.

There was nothing of note in the other abdominal organs, except that the spleen was but slightly enlarged. The heart was normal in conformation; size rather small. Both lungs were much congested at the bases and posteriorly.

There had occurred, then, a perforation which had been walled off and given rise to the first train of uncertain symptoms, while the symptoms immediately preceding her death were due to the fresh perforation above the first. Operation could have reached either perforation readily, as the bowel was in its usual position and the adhesions were not strong enough to have given any trouble. Had it been undertaken with the onset of the first symptoms, however, the second perforation would have occurred subsequent to it, unless the manipulation of the bowel had caused its complete rupture during the operation and its recognition at that time.

The second case was a man, aged twenty-seven years, admitted with the following incomplete history from the family: He had long been much exposed and overworked, and for two months past had had bronchitis. Two weeks before admission he began to feel so weak that he could scarcely do any work, but kept at it until six days before admission, when he went to bed. For several days he had been very delirious. Had had no nose-bleed or diarrhea, but a good deal of cough. Upon admission he was so delirious that he had to be tied in bed, and exhibited all the usual symptoms of profound involvement of the nervous system. The lungs showed only bronchitis, while the heart-sounds and area were normal. The abdomen was moderately distended, there were a few spots; the liver-dulness about normal; the splenic dulness enlarged. There was slight tenderness near the umbilicus. The pulse ranged near 120; the temperature was very variable, from 99° to 103° without baths. When seen for the second time, thirty-six hours after admission, he was evidently in extremis. The pulse was 170; the temperature maintained its irregular course and had shown no collapse fall. The abdomen was very little more distended. But the extremities were cool; he was semicomatose; the liver-dulness was gone front and back, and even in his stupor he flinched on palpation of the abdomen. He died a few hours after. The postmortem showed the following:

Thorax. A slight excess of fluid in the pericardium. The heart moderately hypertrophied; no valvular lesion. The lungs showed a very slight congestion at the bases, and were peculiarly free from pigment.

Abdomen. On opening the abdomen gas escaped and purulent fluid welled into the opening. The omentum and upper portions of the bowel were free from adhesions, while the greater portion of the small intestine was massed together in a lump in the true pelvis, and the loops were strongly adherent to each other and to the pelvic walls, so that it was with considerable difficulty that the mass was released. In doing

so the adhesions between the loops were partly broken up, and a good-sized perforation came into view, which, however, was not itself surrounded by adhesions, but communicated with the general cavity of the peritoneum. Upon opening the bowel the large intestine was found free; while there were a moderate number of ulcers in the last three feet of the ileum. Those near the ileocecal valve had almost entirely sloughed and had clean surfaces; while, as usual, those higher up were less advanced, and some follicles only a little swollen had not ulcerated; 35 cm. from the valve was an ulcer lying across the long axis of the bowel, 4 cm. long and 1.5 cm. wide. About half of the swollen lymphoid tissue had sloughed, while the remainder was still there; very much swollen and very dark, almost gangrenous in appearance, as was the serous membrane beneath. In the center of the ulcer's base was a clean cut perforation of 5 mm. diameter. The other abdominal organs presented nothing out of the usual order.

The matting together of the small bowel in the pelvis, and consequent difficulty in reaching it, and the strength and extent of the adhesions would have prolonged an operation and have required much graver manipulation than in the first case. Further, the extent of the ulcer was too great to allow of turning it all into the bowel lumen in operation, and its base was too nearly gangrenous to have allowed of sutures being taken through it to cover the perforation.

Discussion.—Dr. Daland said that these cases illustrated the great difficulty which the surgeon had in giving relief.

Dr. Hare noted that the perforation came on at the time of a bath; not many cases were recorded in which this occurred. Our present cold-bath treatment he believed to be exceedingly crude.

Dr. D. D. Stewart thought that the reason perforation was not more frequently reported was that the baths were begun early and were more successful, so that perforation did not occur.

Dr. Hughes desired to call attention to two points: First, that perforation usually did not occur suddenly; and second, that perforations were not infrequently multiple. Cases of typhoid fever should be watched, and the surgeon called in time. When shock had developed and the general condition was bad, interference came too late.

Dr. Edsall, in closing, stated that the second case was hopeless when seen; in the first case the symptoms were at first too indefinite to warrant operation. He agreed with Dr. Hare in considering the relation of the bath to the perforation as causal. Abdominal pain and tenderness had been marked, and probably were a danger signal. *January* 28, 1898.

39. *Localized interstitial hepatitis of syphilitic origin.*

Exhibited by J. Dutton Steele, M.D.

The patient was a young unmarried woman, aged twenty-two years, who died from a severe attack of typhoid fever contracted in the hospital. She had been very healthy previously, and never had had any symptoms pointing to hepatic disturbances. She was under the care of Dr. James Tyson, to whom I am indebted for the privilege of examining the specimen.

The autopsy showed the presence of four nodules in the upper and anterior surface of the right lobe of the liver. Three of these nodules measured 1 cm. in diameter, and were slightly raised above the surface of the organ. The fourth nodule was larger, and measured 2 cm. in diameter. The nodules were lighter in color and harder than the surrounding tissue. The surface was smooth, and they resembled secondary deposits of sarcoma or cancer. The typical lesions of typhoid fever were found in the intestine.

Microscopic section shows the raised nodules to consist of areas of chronic interstitial hepatitis. The connective tissue is much increased between the acini, and there are large round-cell infiltration and evidences of proliferation. The liver-cells are flattened and are in different stages of fatty degeneration. The radicles of the hepatic artery show much endarteritis with great increase of the connective tissue of the intima. A close search discovers scattered areas in the newly formed connective tissue, averaging perhaps 50 μ in diameter, which have undergone extensive fatty degeneration. The connective tissue around these areas shows much round-cell infiltration and proliferating changes, and fibroblasts are commencing to push their way into the necrotic material from the connective tissue of the walls. The cause for such a localized interstitial hepatitis was in doubt until the above-mentioned areas of degeneration were discovered, but their presence, associated with the marked thickening of the intima of the arterioles, strongly suggests the idea that they are syphilitic in origin, and that the necrotic areas are merely gummata. There is nothing in the patient's history to suggest specific infection. It is possible that the process was an hereditary one.

February 25, 1897.

40. *Abscess of liver and ulceration of intestines.*

Exhibited by J. Dutton Steele, M.D.

The body is that of an emaciated colored man; there are no marks upon the surface. Upon opening the abdominal cavity the liver projects 6 cm. below the costal margin in the midclavicular line, and 10 cm. in the median line. The gall-bladder is greatly distended, and projects beyond the margin of the ribs. The liver is somewhat adherent to the peritoneum by a thick layer of recent lymph. The liver reaches to the fourth interspace. Upon the left edge of the right lobe, between the costal margin and the ensiform cartilage, there is a softened area giving fluctuation, but not protruding much above the surface of the organ.

The appendix is short, thicker than normal, and appears somewhat cystic. It is slightly adherent to the mesentery, which forms a tight envelope around it. The cecum is somewhat discolored. There are about 500 c.cm. of clear fluid in the abdominal cavity. The pericardium is normal. The pleural sacs are free and normal. The heart is relaxed. The myocardium of the left ventricle is slightly hypertrophied and shows considerable fatty change in the papillary muscles. All the valves and the endocardium on both sides are normal. The lungs show marked hypostatic congestion posteriorly, but otherwise nothing of interest. The spleen is enlarged, and on section the pulp is soft and semifluid. The adrenals are normal. The kidneys are swollen. The capsule strips well. The cortex is anemic, and the pyramids and medullary rays are congested. The cortex has a slight yellowish tinge, and its consistency is lessened. It is lustreless and evidently in a condition of cloudy swelling. The ureters and bladder are normal. The pancreas is normal. The stomach is normal. The duodenum is also normal. The papilla of the gall-ducts is prominent. The gall-ducts themselves are patulous to the end of the common duct. The small intestines show nothing of interest except slight enlargement of the solitary glands.

In the caput coli and appendix are several ulcerations almost round in outline. These show no tubercles on either floor or peritoneal coat. The appendix shows extensive ulceration of its mucous membrane. The ascending colon has four small ulcers. The rest of the colon and the rectum are normal.

The mesenteric glands are not enlarged, but the lymph-glands lying

about the portal vessels are extensively enlarged, softened, and edematous. The common bile-duct is not dilated, nor is the cystic duct. The flow of bile from the gall-bladder is not free. The mucous membrane of the gall-bladder is somewhat swollen and roughened, especially near the opening of the duct, and the bladder is filled in its lower third with a large number of small black gallstones that have exceedingly sharp edges and corners, and appear to be impacted in the mouth of the duct.

The liver is large and of normal color. The lower third of the organ is replaced by a large abscess with rough walls, covered by shaggy lymph. The abscess-cavity is filled with flakes of fibropurulent material floating in a clear serum. The hepatic duct is somewhat dilated and leads directly into the abscess-cavity by its left branch. The right branch is also dilated, and a probe 2 mm. in diameter can be easily passed to the periphery of the liver. In the middle of the right lobe is a small purulent deposit 2 cm. in diameter, but which has no connection as far as can be ascertained with any bile-ducts. The bile-ducts under the surface of the lower part of the left lobe are dilated and plainly visible in branching white lines. A probe can be passed along them and enters into the abscess-cavity. On the right apex of the right lobe is a depressed scar, which is hard and white and somewhat nodular. On section the nodular formation is found to be 7 mm. in diameter.

The portal vein is normal. The portal glands are normal. There is no enlargement or sign of inflammation in the lymphatic vessels. The hepatic artery is normal. The brain and cord were not examined.

March 11, 1897.

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41. *Cylindric epithelioma of the sigmoid flexure.*

Exhibited by James M. Anders, M.D., and Joseph McFarland, M.D.

G. S., aged sixty-five years, by occupation a teacher. The family-history is negative.

Personal history. Enjoyed the very best of health and physical vigor until two years ago. Symptoms of dyspeptic trouble then were manifested. First vomiting occurred about two years ago; constipation was occasional. Has filled his duties as professor in a school until three months ago. During the last three months has had repeated attacks of vomiting, and during the intervals of vomiting had gaseous eructations.

The man seemed to regain his health and strength temporarily, and

would be free from trouble for a week at a time. At least six weeks before his death he did not have a normal passage. Only occasionally, probably once a week, the enemas were accompanied by any fecal matter. There was no special wasting until within the last two weeks. Throughout the whole illness there were slight febrile changes.

An abdominal section showed a neoplasm at the sigmoid flexure. It consisted of an annular constriction of the gut, whose wall was greatly hypertrophied. The tumor was removed and Murphy's button introduced. The patient died.

Upon microscopic examination the tumor proved to be cylindric epithelioma. *March* 25, 1897.

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42. *Series of diseased stomachs.*

Exhibited by ALFRED STENGEL, M.D.

CASE I. *Cyst of the Stomach.*—The specimen I show was obtained at the autopsy upon a case that had presented no gastric symptoms of any kind. The postmortem examination revealed renal lesions presenting no peculiar features; there was also atheromatous disease of the aortic valves and general atheroma, most marked in the splenic and other abdominal vessels. The feature of interest, however, was a curious tumor connected with the stomach, which is the specimen shown. This was not discovered until the body was opened. The stomach was displaced downward, and the lesser curvature alone was visible, the tumor occupying this situation. It was evidently cystic, translucent in character, and contained serous liquid. In size it was about 7 or 8 cm. in diameter. The tumor could be shelled out from beneath the layers of the gastrohepatic ligament, and it had apparently formed between the two layers of this peritoneal reflexion. The walls were quite thin. From the outside the appearance suggested loculation, partitions seeming to pass from the walls to the center, but on section there was found a single cyst. The liquid within was serous in character, and coagulated at once on removal. The only explanation of the nature of this tumor I can offer is that it was a cyst following a hemorrhage, though there was no clinical history pointing to such an occurrence. We might have expected sudden vomiting, pain, or other gastric symptoms of a severe kind. The tumor is most unusual, and I know of but one similar case, that reported by Ziegler in the course of the current year. In that case the tumor was much larger and presented much thickened walls. In addition it was

situated beneath the serous coat of the stomach itself. Aspiration of the tumor was performed twice.

Case II. *Cirrhotic Carcinoma of the Stomach; Dilatation and Ulceration of the Esophagus.*—The patient was a very old man, who gave a history of gastric trouble extending over a number of years without any marked signs pointing to carcinoma. There was no dilatation of the stomach discoverable clinically, and there had been no vomiting. The history was that of chronic gastritis. The autopsy showed an unusually small organ which scarcely held a pint of liquid. The walls of the stomach were enormously thickened and presented irregular elevations on the surface. The disease involved the organ from the pyloric end to the cardia. There was no actual obstruction of the latter, but the general thickening of the walls was most marked at the cardiac end, and this doubtless caused some impediment to the passage of food. The esophagus was greatly dilated, and at the autopsy was distended with material looking like curdled milk. It was fully 10 cm. in diameter, and presented on the mucous surface a number of deep ulcerations, one of which ruptured as the organs were removed. The condition is simply one of uniform dilatation of the esophagus, with secondary ulceration due to an unusual form of obstruction. As far as the stomach is concerned, I should like to call attention to the fact that many of the older reports of cirrhosis of the stomach were probably of this character, that is to say, carcinoma with contracture. The microscopic examination in my case showed it to be undoubtedly carcinomatous, with enormous thickening of the connective tissue as well.

Case III. *Cirrhotic Carcinoma of the Stomach.*—The stomach I now exhibit presents no important features excepting contracture and thickening of its walls. Microscopically it is like the last one, cirrhotic carcinoma.

Case IV. *Marked Dilatation of the Stomach with Thickening of the Walls.*—The clinical history of the case from which this specimen was obtained is unknown. The stomach is enormously enlarged, and would doubtless have held during life two or three quarts of fluid. The cause of the dilatation is evidently the thickened fibrous ring at the pylorus. The walls of the stomach are greatly thickened, and as far as the naked-eye appearances are concerned it would be difficult to distinguish this from the two previous cases. Sections, however, taken from various parts of the organ have been studied, and these show great hyperplasia of the connective tissue of the submucous and the serous coats, with some muscular hypertrophy and intermuscular fibrous overgrowth. In other words, there is uniform fibrous thickening of the organ.

Case V. *Beginning Ulceration of the Stomach.*—The specimen I now show presents a beginning ulceration at the cardiac end in the anterior wall. On the opposite wall of the stomach is seen a similar lesion, which, however, is almost completely healed. Clinically it is of interest that this patient presented no gastric symptoms of any kind. He died of general arterial disease with nephritis. In the fresh state the ulcer presented itself as an area of erosion with numerous dark-red, slightly elevated spots in its floor. These somewhat resembled enlarged and hemorrhagic papillæ or loops of bloodvessels, greatly distended and projecting somewhat like a knuckle over the surface of the erosion. It was, therefore, quite different in appearance from an ordinary ulcer. Its mode of formation is somewhat obscure. The association of renal disease is of interest in connection with certain statistical studies lately made by English physicians, which tend to show that renal disease and ulceration of the upper part of the intestinal tract (principally duodenum) are frequently associated. Whether there is any etiologic relation in this case is doubtful. The case is also interesting from the absence of clinical symptoms. I have long believed that slight erosions of the mucous membrane of the stomach are more common than we are disposed to think. I should not be surprised if it were true that many attacks of slight indigestion, of colic, and the like are accompanied by erosions of the surface. I know that the claim has been made by certain specialists that such erosions do actually occur, and the attempt has even been made to draw a clinical picture of such cases. Unfortunately this is not at the present time feasible.

The instance under discussion to-night is rather more than a simple erosion. There has evidently been a distinct vascular disturbance, with secondary erosion. I cannot, however, pretend to give a satisfactory explanation of the origin of the whole process.

Case VI. *Diffuse Myoma of the Stomach.*—The specimen I now present is one of very unusual interest to me, and as far as I know has no counterpart in literature. The specimen was exhibited to the Society at the time of its removal, a year or two ago, and before accurate studies could be made of it. It presented at that time, in its fresh state, somewhat the appearance of diffuse colloid carcinoma. The wall of the organ was uniformly thickened and gelatinous in appearance. More careful examination, however, shows that the mucous membrane is apparently healthy, even to the naked eye, and the thickening of the walls is due to great increase in the muscular coat. On microscopic examination there is found enormous hyperplasia of the muscular layer and some increase of smooth muscle in the submucous layer as well. Every part

of the stomach is involved, from the cardia to the pylorus, and the walls are uniformly about 1½ to 2½ cm. in thickness. The rugæ of the organ are exceedingly prominent and the pyloric ring is hyperplastic, though there is no distinct pyloric obstruction. During life the patient suffered with gastric symptoms, and an exploratory incision was made; of course, without any result.

I know no other designation to give this condition but diffuse leiomyoma of the stomach, and, as I stated in the beginning, I have not been able to find reports of a similar case in literature. In connection with this specimen I wish to call attention to the localized nodular myoma which I previously presented to the Society, and the report of which appears in the last volume of the Transactions. *December* 12, 1895.

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43. *Stomach from a case of rumination.*

Exhibited by David Riesman, M.D.

This stomach comes from the case of rumination whose suprarenal capsule I have described this evening, and whose history I have reported elsewhere. No macroscopic lesion was found that could explain the rumination. This has been the result of most autopsies in cases of merycism. Arnold and Luschka found dilatation at the lower end of the esophagus and an unusual thickness of the internal branch of the spinal accessory nerves, the branch that unites with the vagus. As in ruminants the gullet terminates in a funnel-shaped expansion, and as in them the internal branch of the spinal accessory nerve is very thick, Arnold, and also Luschka, concluded that the affection was accounted for by these lesions. But subsequent studies have not confirmed this, and as far as the dilatation of the esophagus is concerned it is not present in this instance. *March* 25, 1897.

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44. *Intussusception in an infant fifty-four hours old.*

Exhibited by David Riesman, M.D.

This specimen of intussusception of the intestine, which I am permitted to show through the kindness of Dr. B. C. Hirst, comes from a colored female infant, born in the University Maternity, at 12.08 A.M.,

February 14th, 1897. The mother was in labor, which was not difficult, about twelve hours. The child breathed once after the head was delivered and before the body was expelled. After birth it cried lustily, and seemed perfectly well until 8 A.M. of the following day, when the temperature rose to 104° F., respiration being 50, pulse 180. At midnight a rather copious discharge of bloody fluid and clotted blood occurred from the bowel. Up to that time the infant had had one normal discharge of meconium and one free brown movement. At 2 A.M. it vomited a brownish material, and died at 6 A.M., fifty-four hours after birth.

Autopsy. The body is 48 cm. long; the umbilical stump is still present, but is dry and mummified. From each breast two or three drops of a turbid green fluid can be expressed, which on microscopic examinatoin consists of fat-globules and granular corpuscles.

An *intussusception* exists in the region of the jejunum. Above the intussusception the intestine is distended, the serous coat intensely injected and in places hemorrhagic. At the seat of the invagination the gut is almost black from hemorrhagic infiltration; below this point are numerous hemorrhages involving the peritoneal coat and varying in size; in places they extend around the entire circumference, in others part way, in still others they are discrete, the size of a pin's head; they diminish in extent with increase in distance from the lesion. The intussuscepted part consists of three spirally arranged, closely approximated black coils, thickened and very firm to the touch; the outer layer of bowel slips easily over the inner. The mesentery shows small hemorrhages, and its lymph-glands are much enlarged.

We are evidently dealing here with an antemortem, pathologic, or obstructive (Treves) intussusception, which had produced strangulation, with intense congestion of and hemorrhage into both the intussuscipiens and the intussuscepted part of the bowel, and hemorrhages of less intensity into the distal part of the small intestine Had the child been older it is possible that sloughing of the bowel would have occurred, leading perhaps to spontaneous recovery. There are no inflammatory adhesions between the outermost coat and that next to it. The intussusception naturally exerted an influence on the mesentery which at its entrance into the intussuscipiens was twisted and greatly compressed. The position of the invagination is somewhat unusual, as 70 per cent. of all intussusceptions in children are ileocecal.

As to the pathogenesis of the process, we may accept the view of Nothnagel, which is endorsed by Treves and D'Arcy Power, namely, that one piece of bowel by the activity of its muscular coat (probably the longitudinal fibers) is drawn upward over a contracted more

proximal part. As D'Arcy Power remarks: It is due to one piece of gut swallowing, so to speak, a neighboring piece, which has become constricted in consequence of a vigorous contraction of its circular coat. Both Nothnagel and Power believe that intussusception is spasmodic in origin, and not, except in rare instances, paralytic.

The exciting cause of the intussusception in this case has not been determined. The condition may have been, and this view appears to me most logical, the result of an overactive peristalsis, during which one part of the bowel was momentarily more strongly contracted circularly and was promptly "swallowed" by the part beyond.

A polyp in the bowel might have acted as the exciting cause, and, as the intestine has not been incised, such a possibility cannot be excluded; but it is scarcely likely in so young a child, nor do inspection and palpation afford any ground for such a suspicion.

The case is probably one of the youngest on record, and if we assume, as I think we may, that the occurrence of the intussusception coincided with the rise in temperature, the child was really only thirty-two hours old when the intussusception took place. *March* 25, 1897.

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45. *Secondary sarcoma of the duodenum.*

Exhibited by Joseph Sailer, M.D.

The specimen that I present to-night has been taken from the duodenum of the boy whose brain and kidneys I presented at the Society a few weeks ago. When the specimen was removed it was observed that there were a number of small nodular elevations upon the surface of the mucous membrane. These were of firm consistency, and in the fresh specimens slightly paler, but the color-differentiation disappeared during the hardening in Müller's fluid. Transverse section revealed the following characteristics: serosa was normal; muscularis was normal; the submucosa and the mucosa showed the following alterations: at the point where the nodules were most prominent was found a mass of small, round or oval cells, staining deeply with hematoxylin, and the other nuclear stains at the periphery, but toward the center the color was not so well taken, and differentiation was far from perfect. Little masses of these cells stained faintly with eosin. Round some of the large nodules there was a distinct layer of cells, sharply divided from the others, whose

nuclei stained even more deeply, and which resembled the ordinary round-celled infiltration, particularly such as one finds in chronic inflammatory processes. In some of these masses of cells bloodvessels were observed, whose walls were composed of a single layer of endothelium, and in a few places there seemed to be a slight interstitial hemorrhage. Besides the nodules there were scattered throughout the mucosa particularly, and often nearest the periphery, collections of round cells, similar in every respect to the more circumscribed masses. The glands of the intestinal tract were normal, the nuclei staining especially well. The bloodvessels showed no pathogenic alterations. In regard to the nature of these cellular collections, it seems to me that there could be but two conditions giving rise to such an appearance—either hyperplasia of the lymphoid elements, or secondary sarcoma. The situation of lymphoid follicles in the duodenum corresponds with the position of these nodular outgrowths; although in general it can be said that the collection of lymphoid cells is more extensive in the submucosa than in the mucosa; whereas in the present instance exactly the reverse is the case. The lymphoid masses are much more discrete and show less tendency to infiltration. They rarely, even under exceptional circumstances, show degenerative changes when the rest of the mucous membrane is intact. Bloodvessels when they exist are normal in character. Hemorrhage occurs only in acute infectious diseases. In the present instance, besides the nodules, there is considerable cellular infiltration of the mucosa, and it is to be observed that the cells are sometimes oval in shape, a condition that is rare in lymphocytes. Moreover a few bloodvessels have been found that resemble the sarcomatous blood-spaces, and as the chief point in the distinction of the question we have the existence of multiple primary sarcomata in the kidneys. It may be urged that there is no apparent metastasis to the other organs. It is difficult to understand why it should be limited to the duodenum, and I confess myself unable to explain this peculiarity; especially as the renal blood supply is not connected with the portal system. Nevertheless I am inclined to regard these nodules as sarcomatous in nature. A point against sarcoma that is of considerable weight is the absence of invasion of the vessel walls by cellular infiltration. *April* 8, 1897.

46. *Endothelioma of the omentum.*

Exhibited by S. W. Morton, M.D.

The specimens I am about to present are of interest not only on account of the rarity of this form of malignant growth, but because of difference between the symptoms observed in this case and those of the more common form of malignant disease of the abdomen.

The history of the case is as follows:

Mary D——, Asbury Park, N. J., aged fifty-five, single, teacher by occupation. Her father died when past seventy years of age of "dropsy of the chest;" her mother, when about the same age, of chronic diarrhea; two sisters died of malignant disease, one of cancer of the stomach, the other of a malignant tumor of the abdomen, similar in its symptoms to this case. Another sister died of influenza, and three are still living in apparent good health.

Our patient had the usual diseases of childhood but no other illness until this fatal disease began. Her life, as is usual with those of her vocation, was not conducive to health. She took little out-door exercise, and had poor and possibly insufficient food. She was noticed by her friends and relatives to be failing in health for months before she gave up her teaching. She had been growing gradually weaker and more emaciated; had little appetite, and suffered constantly from obstinate constipation. Her physician, Dr. Sarah F. Mackintosh, of Asbury Park, has since confirmed these statements, and adds that there were in addition symptoms of atonic gastric dyspepsia with flatulence, and that she was inclined to view the case as one purely of that character until a tumor was discovered, when the idea of malignant disease presented itself.

In June, 1895, our patient was forced to give up her school. Rest from her work made no favorable change in her downward progress. In October she sought relief in St. Luke's Hospital, New York, where she was informed that her case was not an operative one. October 31st she was admitted to the cancer-ward of the Philadelphia Home for Incurables. At that time she complained of great weakness, anorexia, and constipation.

She was of small stature and delicate frame; had the appearance of a very sick person; was much emaciated; abdomen rather prominent in comparison with other portions of body. Skin was cachectic in appearance; mucous surfaces pale but not icteroid. Heart, lungs, liver, and spleen apparently normal. Stomach and bowels could not be outlined.

Vaginal examination was not permitted. An extensive, dense tumor extended from high up in the epigastrium and hyperchondriac regions to two inches below the umbilicus. Its surface was flat, slightly irregular or roughened, but not presenting any distinct elevations or depressions. It was slightly movable, but did not move with the diaphragm, nor did it seem to be connected with the liver or spleen. The lower edge was rounded and presented a slight curve from one side of the abdomen to the other, but was not notched. There was no especial tenderness, and, as you will have observed, there was no pain complained of at any period of the disease. No evidence of fecal impaction; no tympanites. Could not detect any effusion; no eructation of offensive gases or fluids, but breath had a fecal odor.

Urine, sp. gr. 1018. Examination was negative.

She remained with us for about six weeks, meanwhile growing more and more weak. During all this time she continued to be quite constipated. During the last two weeks her mental condition was not clear, and some edema occurred in the feet, and effusion into the abdomen. She died December 18, 1895, apparently from sheer exhaustion.

Autopsy, December 19, 1895. *Thorax.* Heart, pericardium, lungs, pleuræ, aorta, and mediastinal glands were all examined carefully, and found to present only the atrophy consequent to general failure of nutrition which accompanies malignant disease.

Abdomen. Walls thin; fat scarcely discernible; not quite sufficient sanguineous serum present to overflow from the median incision. Parietal peritoneum was everywhere studded with small, whitish, hard growths from the size of a mustard-seed to that of a hazel-nut. In the bottom of the opening lay a flat, hard mass honeycombed with openings of varying sizes from that of an ordinary pin-hole to one one-third of an inch in diameter. This mass hid from view all other abdominal organs except the liver. It was movable; two to two and a half inches in thickness, by eight to ten inches in breadth and length. When lifted it was found to be rather closely attached to the stomach and intestines, the latter being agglutinated by numerous abnormal adhesive bands into a mass which could not be easily unravelled. In places they were attached to the parietal peritoneum, so as to make it difficult to sever this connection without the aid of a knife. There was everywhere where the peritoneum could be seen the same character of little growths as I have described as being on the parietal peritoneum, *e. g.* the peritoneal coverings of the liver, the bowels, spleen, and even down in the pelvis, a condition much in appearance like "carcinomatosis." The stomach and intestines, with the exception of the peritoneal coverings, were not

involved. The liver was in the same condition, it showing on section no evidence of special disease or new growths. Have not made an examination of the kidneys and pelvic organs, but as far as I could discern with the naked eye they had not been involved by this peculiar new growth. It was plainly evident that the large, flat, dense, honeycombed mass above referred to was a diseased omentum.

No examination was made of the head.

The specimens I present to-night were sent at once to Dr. John Guitéras' Pathological Laboratory, where sections were made by Dr. Joseph McFarland. I am enabled, through the kindness of these gentlemen, to present to you this evening a microscopic section of the omental growth, which has been diagnosed by Dr. Guitéras as an endothelioma.

Dr. McFarland has, I believe, presented specimens similar to these on one or two occasions, and is, I believe, better able to treat of the histology of these tumors than I, and shall be pleased to hear him upon the subject of "endotheliomata." *May* 13, 1897.

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47. *Cylindric epithelioma of sigmoid flexure.*

Exhibited by J. J. Rectenwald, M.D., for Dr. D. Riesman.

The patient, white, sixty-three years old, was sent from the Blockley outwards to the Philadelphia Hospital. The history is as follows:

Ten years ago had an apopleptic seizure following a debauch; was an excessive drinker of whiskey. For the last nine months he had been complaining of weakness and constipation, but was able to be about and enjoyed rather good health until three months ago, when he complained of severe abdominal pain, tympany, and constipation, for which he was treated, in the outwards, by rectal injection, and in a few days was able to be about again. For the last three weeks was constantly taking laxatives; but continued his work in the blacksmith-shop until within two days of his being sent to the medical wards. In the outwards he had been given several enemas which did not return. On admission to the hospital he was found to be a very stout man, weighing over two hundred pounds; features pinched; temperature subnormal; abdomen greatly distended and tympanitic, but not very tender; perineum swollen and edematous; rectum slightly prolapsed. His bowels had not moved for three days; an enema of two pints was given, which did not return. Upon the injection not returning it was surmised that a lesion existed

low down. He vomited twice during the first day in the hospital, but did not vomit any after that. He died April 29, 1897, after being comatose for a few hours.

Postmortem: In the sigmoid flexure, eighteen inches from lower end of bowel, is a contraction through which water passes with difficulty, there being almost complete obstruction. Nothing striking is found on the exterior of the bowel at this point to indicate its condition in the interior other than retraction of the fatty tissue at this area. Upon opening the constriction it is found to be an ulcer, running transversely to the axis of the bowel, and measuring 26 mm. in height; the edges are ulcerated; the base of the ulcer is depressed and cicatricial. At one part is a perforation the size of a pin-head; this leads into a pocket the size of a large pea, the wall of which is composed of adherent mesenteric fat. The floor of the ulcer feels hard and fibrous. Dr. Riesman at the time thought it was a cylindric epithelioma. The portion of intestine below this shows follicular enteritis. Above the large ulcer are several small ulcers with rounded edges. The rectum has a funnel-shaped termination, large end down.

The kidneys were the seat of chronic interstitial nephritis.

Microscopic examination made by Dr. Riesman showed growth to be a cylindric epithelioma. *May* 13, 1896.

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48. *Primary carcinoma of the pancreas, with extensive metastasis.*

Exhibited by J. P. Arnold, M.D.

The specimens presented are from a case which died in the wards of the Presbyterian Hospital a few days ago.

The patient was a woman, aged sixty-one years, admitted to the hospital with a history of having lost flesh for the past nine months. Two months ago was taken with severe pains in the abdomen, and since that time has failed rapidly. She complained of great weakness, dyspepsia, and constipation.

The family history was negative.

The woman was extremely emaciated and cachectic. Mental condition very poor; she was very irritable and restless, making a physical examination almost an impossibility.

The following notes were made on the examination of the abdomen: Skin dry and wrinkled; walls thin; the abdomen is distended and

tympanitic, especially in lower half. The veins are prominent; walls not tense; movable dulness in the flanks; liver-dulness in the midclavicular line, from sixth rib to within 3 cm. of transverse umbilical line. Edge distinctly palpable. There is a small nodule, size of pea, in right epigastrium near costal margin. In the right midclavicular line, just above the line of the umbilicus, is felt a nodular mass irregularly oval in outline, 7 x 4 cm., which descends about 2.5 cm. with inspiration; it is not distinctly tender nor movable. Attempts at a more complete physical examination made impossible by patient's objections.

The woman gradually became more and more toxemic, and died in two weeks after admission. The stomach-contents could not be obtained. There was no jaundice or glycosuria, nor were the stools fatty.

Autopsy. Autopsy, twelve hours after death. Body of an extremely emaciated white female. Postmortem rigidity slight. There are old pleuritic adhesions on both sides anteriorly. Right pleura contains 250 c.c. clear fluid. Pericardial sac normal. Heart in systole. A moderate degree of thickening of mitral valve, and some atheroma of aorta. The right lung is tightly adherent to the diaphragm, and presents in the lower lobe numerous, scattered, small, hard nodules, varying in size from a shot to a small pea. Section shows the nodules to be white and fibrous.

Abdomen. Peritoneal cavity contains 1000 c.c. of fluid. Liver extends in right midclavicular line to level of umbilicus, in median line 7 cm. below xyphoid, and is at the costal margin in left midclavicular line. The mesentery covers small intestines, and presents numerous, small, hard nodules, size of a pea.

Above and behind the pylorus, in the region of the head of the pancreas, is a hard spheroidal mass with a nodulated surface the size of a tangerine orange. The liver is tightly adherent to the diaphragm, and laterally to the parietal peritoneum. It presents numerous yellowish-white, irregularly nodulated elevations, varying in diameter from 1 cm. to 4 cm.; some of these nodules have a depressed center. The gall-bladder extends a short distance below the edge of the liver. Its peritoneal coat is somewhat roughened. The stomach is small and apparently free from new growth. The duodenum is dilated. The small intestines present a few nodules of new growth, especially in the lower part of the ileum. Some nodules are also seen in the large intestine. The mesenteric glands are enlarged and hard.

Spleen adherent above. One large nodule.

The pancreas. The head is everywhere adherent to the surrounding tissues, from which it cannot be completely separated. It presents a

nodular mass of new growth the size of a small orange. The tail is apparently not involved.

Diaphragm adherent to the structures above and below and much infiltrated.

Kidneys rather paler than normal. Cortex yellowish. Uterus and adnexa not affected.

Sections of the pancreatic growth, of nodules in the lung, liver, and spleen, and of a mesenteric gland, examined microscopically, show the growth to be a scirrhous cancer. The section from the pancreas showed a great excess of stroma. The single growth in the pancreas, with its large size and general characters, led me to believe it to be the primary lesion.

The points of interest in the case are the absence of jaundice, vomiting, fatty stools, and glycosuria. Nor were there signs of obstruction to the pancreatic duct. The presence of ascites would seem to have been due to pressure upon the portal vein. The occurrence of a right-sided hydrothorax is of interest in view of the recent paper of Dr. J. D. Steele on "Cardiac Hydrothorax." *May* 27, 1897.

III. THE CARDIOVASCULAR AND HEMAPOIETIC SYSTEMS.

1. *Tricuspid and mitral endocarditis.*

Exhibited by Frederick A. Packard, M.D.

K. K., a white female child, aged about ten years, was admitted to the Children's Hospital on April 16th, and died on June 16, 1895. For a year before admission she had had frequent attacks of rheumatism, but until two months before admission these had not been very severe. She then had an attack so intense as to cause her to go to bed for about six weeks. She had been out of bed, although complaining of precordial pain, for the three weeks preceding her admission. Examination on admission revealed a double apical murmur with symptoms and signs of broken compensation. She improved under treatment so that by the latter part of May she was sent to the Country Branch.

On June 9th she was sent back to the hospital with intense edema of the whole body, except the back. There was a quivering pulsation over the whole precordium and in the epigastric region. The veins of the neck pulsated violently, and those of the surface of the thorax were plainly dilated. Palpable pulsation was present in the epigastrium. The apex-beat was felt in the fifth interspace in the nipple-line and somewhat to its left. At the apex there was a smooth, rather prolonged, systolic murmur preceded by a shorter, but also smooth murmur. At the xyphoid only the systolic portion was heard, and this could not be separated from that at the apex. At the aortic area there was an almost inaudible systolic murmur which was not transmitted. The second sound at the aortic area was very feeble. A faint systolic murmur was heard at the pulmonary area, where also the second sound was accentuated. Cardiac dulness began at the third rib and at the midsternum.

The heart's action steadily failed, edema increased, the urine became very albuminous, and death occurred on June 16, 1895.

At autopsy there were found much subcutaneous edema; a large amount of fluid in the peritoneal cavity; double hydrothorax; much clear serous fluid in the pericardial sac, which also showed beneath the visceral layer many small ecchymotic spots of bright red color; marked

edema of the lungs; slight splenic enlargement; kidneys engorged; liver congested and heavy. The heart presented to the Society weighed 240 grams. The right heart was tremendously distended with dark fluid blood, the left with fluid blood and currant-jelly clots. In the auricular appendages there were white, firm, and adherent clots. The whole heart was of globular shape. The tricuspid orifice was of normal size; on each tricuspid leaflet there were a few small pinhead-sized vegetations of yellowish-white color on the auricular surface just within the free edge. The mitral valves were slightly thickened, but the endocardial covering was smooth and normal. The aortic leaflets were smaller than the pulmonary, but both arterial valves were normal. The wall of the right ventricle measured 3 mm. in thickness, that of the left 10 mm. Foramen ovale and ductus arteriosus closed. *October* 24, 1895.

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2. *Peri- and endocarditis, the latter involving both sides of the heart.*

Exhibited by Frederick A. Packard, M.D.

The case from whom this specimen was taken was a boy, aged eleven years, who was admitted to the Philadelphia Hospital on April 17, 1894. He came from a society for assisting poor children, and no previous history was obtainable. During life he had great dyspnea, restlessness, and precordial pain. Examination of the heart revealed great heaving of the whole precordium; an apex displaced far downward and to the left; an area of cardiac dulness extending from the lower border of the second rib and from the right border of the sternum; a faint thrill near the apex; creaking friction-sounds, and a loud double murmur, having apparently three points of greatest intensity—at the mitral, aortic, and tricuspid areas. Direction of transmission of the murmurs could not be very definitely determined, owing to noisy breathing, dyspnea, and great restlessness. However, a distinct transmission of one of the mitral murmurs to the left could be differentiated, and he had a distinct capillary pulse in the finger-nails. On the day before his death systolic retraction at the apex could be demonstrated, and on the same day he developed pleural friction in the left axillary region. He died ten days after admission.

At autopsy there were found recent pleural adhesions, readily separated, on the left side. The pericardium at first seemed universally adherent, but after cutting through the thick parietal layer a finger could be

inserted, and readily broke through the connecting adhesions. Both visceral and parietal layers were covered with shaggy lymph.

Both lungs were much congested, but showed no areas of consolidation or infarction. The spleen was large, weighed four ounces, was firm, and of a deep currant-jelly color. The liver was enlarged, congested, and weighed $34\frac{1}{2}$ ounces.

The heart weighed $13\frac{1}{16}$ ounces after dissection. The whole surface was covered with rough, shaggy lymph, especially thick on the anterior surface. The apex was made up of the left ventricle. The right auricle was dilated, the wall with its thickened pericardium measuring 8 mm. in thickness. Upon the auricular surface of the anterior leaflet of the tricuspid valve, just within the free edge, there were a few bead-like vegetations of about the size of a pin's head. The endocardium of the right ventricle was slightly opaque. The foramen ovale was completely closed. The cavity of the left auricle was somewhat dilated. Around the entire margin of both leaflets of the mitral valve there was a line of vegetations projecting beyond the attachments of the chordæ tendineæ; these vegetations were quite erect and averaged 1 mm. in height. The chordæ tendineæ were thickened and white. The undefended space was fully closed. The left ventricular wall measured 11 mm. in thickness. The general endocardium of the left ventricle was thickened and opaque. The aortic leaflets were thickened, somewhat rigid, opaque, and showed pinhead-sized vegetations along and close to the free borders. *October* 24, 1895.

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3. *Tricuspid and mitral stenosis.*

Exhibited by FREDERICK A. PACKARD, M.D.

The specimen was removed from the body of a young woman, admitted to the Pennsylvania Hospital on March 28, 1893, with total loss of power in the left arm and leg, and partial palsy of the left face, of sudden onset. On admission she was in profound collapse, but reacted by the next day. Examination of the heart during life was extremely unsatisfactory, owing to the large bubbling râles over both lungs and her noisy respiration. It was possible, however, positively to detect a double murmur near the apex, but its time, point of maximum intensity, and direction of transmission could not be definitely determined. The urine was loaded with albumin, and contained many hyaline, pale, and dark granular casts and, at times, much blood. She was desperately ill

during her whole stay in the hospital, but lived for two weeks with almost no change in her symptoms and signs, except those of growing weakness.

The heart weighs 17 ounces. The right auricle shows some rough organized lymph on its exterior surface. The right auricle is tremendously dilated, and its wall is thickened to a full quarter of an inch. Marantic thrombi are found adherent to its wall. The tricuspid valves are welded so as to form a funnel, the base of which just allows the tip of the middle finger to engage. The right ventricle is dilated and hypertrophied, many marantic thrombi being lodged in its recesses. The left auricle is dilated and hypertrophied, and contains an adherent polypoid thrombus attached to its posterior wall. The mitral orifice is represented by a mere slit at the bottom of a funnel-shaped passage that will not admit the tip of the index finger. The mitral valve leaflets are welded together and of cartilaginous hardness. There is some opacity of the endocardium in the neighborhood of the undefended space. The aortic leaflets are thickened and rough, but not calcareous.

The lungs contain a few recent infarcts, as do also the spleen and kidneys. *October* 24, 1895.

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4. *Verrucose endocarditis of the tricuspid valve.*

Exhibited by J. Dutton Steele, M.D.

Female, aged twenty-eight years. No hereditary disease. Patient had had no previous attack of rheumatism. No specific history, and no evidence of infection upon admission. The attack of rheumatism began suddenly August 13, 1896, after a cold bath, and was polyarticular in type. Upon admission she had a systolic murmur at the apex, transmitted to the axilla, and heard loudest in the third interspace to the left of the sternum. The murmur was soft and low-pitched, and was probably hemic in origin. During the next ten days the murmur grew louder; the first sound at the apex lost its muscular tone, and the apex rose to the fourth interspace. She was slightly delirious. Her temperature ranged from 99° in the morning to 102° in the evening, twice going to 104°.

On August 26th there was a distinct erythematous eruption over the whole body. The aortic and pulmonary second sounds were accentuated. August 30th a galloping friction developed one inch to the left of the sternum in the third interspace and also in the second inter-

space, with the impulse in this position. On September 9th signs of pericardial effusion appeared, and also there appeared to be some fluid in the left pleura. The effusion in these areas increased. Edema of the back and feet appeared, and the patient gradually sank until death, which occurred on the 6th of October.

The autopsy was performed by Dr. H. W. Cattell. There was marked edema of the thoracic walls posteriorly, and of the abdominal walls of the thorax on the left side anteriorly, extending up to the neck. There was a moderate amount of straw-colored fluid in the abdominal cavity. The left pleural cavity was full of a similar fluid. The right pleural cavity also contained a moderate amount. The pericardium contained 32 ounces of a mucilaginous fibrinopurulent material. It was pale-yellow in color, and there were greenish flakes and large masses of yellow fibrinopurulent material floating in it. The apex was bound to the parietal layer of the pericardium, posteriorly, by quite firm adhesions. There were no other adhesions in the pericardium. There were adhesions between the pericardium and pleura on both sides. The pericardium was uniformly thickened, and was dense and white. Both the parietal and visceral layers showed numerous superficial ulcers.

The left lung was entirely atelectatic, and pressed upward to the apex of the pleural sac. It weighed 120 grms. The right lung weighed 495 grms., was congested and edematous, especially at the base.

The liver, spleen, and kidneys were cyanotic. The heart and pericardium together weighed 690 grms. There was a large black clot in the left ventricle. The mitral valves were normal. The aortic and pulmonary valves were normal. There was a large patch of vegetation at the base of the posterior tricuspid leaflet, on its upper surface. The endocardium of the valve had lost its lustre, and was red and swollen, but there was no evidence of previous disease. The myocardium was pale and glistening and seemed to be in a condition of parenchymatous degeneration.

Microscopic examination of the vegetation showed it to be a mass of fibrin closely adherent to the valve. There was much round-cell infiltration of the valve and proliferation of the connective tissue that in several places was commencing to push its way into the thrombus. There were masses of cocci around the periphery of the vegetation, and in a few places in the tissue of the valve itself. These stained by the ordinary aniline dyes, and did not decolorize by Gram's method.

October, 1895.

5. *Ulcerative endocarditis.*

Exhibited by H. M. Fisher, M.D.

This specimen is from a patient who was operated on for appendicitis last June, when the appendix was removed. He was admitted to the hospital again three weeks ago, when he stated he had been perfectly well since the operation, but was then complaining of abdominal pain. He had continued fever while under my care, with marked exacerbations, and the diagnosis of a septic process was made. The specimen shows a very interesting vegetation of the aortic and mitral valves. Both show this; and the mitral has been ulcerated through. The pancreas was very much indurated and presented minute infarctions on microscopic section. The spleen also showed numerous infarctions, as did the kidney and some of the other organs. I think this was a case of ulcerative endocarditis. *November* 14, 1895.

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6. *Malignant (gonorrheal) endocarditis, with congenital anomaly of mitral valve.*

Exhibited by Alfred Stengel, M.D.

The following case came under my own observation postmortem. The history and postmortem study are given as minutely as possible.

Lillian D., a young woman, aged twenty years, was brought to the German Hospital October 12, 1895, and was placed under the care of Dr. Lawrence Wolff, to whom I am indebted for the permission to publish the case. The following history was obtained:

The patient's father died of phthisis at thirty-four years. Her mother is still living and well. The patient had neither brothers nor sisters.

She herself had most of the diseases of childhood, including scarlet fever, measles, diphtheria, and mumps. At the age of seven she had a severe attack of inflammatory rheumatism, and is said to have developed "heart-disease" at that time. The illness lasted about two months, and the convalescence was slow and protracted. About two years later she was ill again, and was told that she had typhoid fever,

but the illness lasted only two weeks, and she does not now recall any of the symptoms.

From this date (when she was nine years old) until the present time she has been well, excepting that her heart has troubled her greatly at times. Palpitations and dyspnea were frequent and easily provoked, but she never suffered with marked rupture of compensation.

A year before her admission to the hospital the patient had begun to have intercourse with a man, and soon developed a distinct and characteristic discharge. She was treated for this, but the discharge continued, and was still present when she entered the hospital.

Six days before her admission to the hospital the patient became ill, the onset being rather sudden. The first symptom was diarrhea, but tympanitic distention of the abdomen and pain over the heart promptly followed. She suffered with headache, vertigo, and a tendency to syncope on slight exertion. Her appetite was not much impaired, but vomiting interfered with the taking of food. The abdomen was uniformly, though only moderately, distended; the pain was diffuse. There were from two to five loose, yellow stools daily; and the patient complained of slight burning pain on urination. The cardiac pain was constant, but not severe. The pulse was weak and rapid; the rate was 124 per minute. Temperature, 38.5° C. (101.2° F.).

Physical Examination. The patient was quite prostrated; dyspnea was evident. The apex-beat of the heart was found in the seventh interspace and outside the nipple-line. It was vigorous and fairly regular. On percussion the cardiac outline was found enlarged, mainly to the left and downward. Auscultation discovered peculiar cardiac sounds. In the sitting posture a loud and distinctly marked presystolic murmur was heard over the apex, followed by a less distinct systolic murmur. Another systolic murmur was audible in the aortic region. The pulmonary second sound was accentuated. In the recumbent posture the presystolic murmur was scarcely or not at all audible, but the systolic murmur could be heard at the apex and as far to the left as the axilla or angle of the scapula.

Examination of the lungs revealed no abnormality. The abdomen was moderately distended; the skin was normal (no eruption); there was tenderness on palpation, but nothing abnormal beyond this. The belly was tympanitic, and the area of splenic dulness somewhat enlarged.

The patient was depressed. Reflexes, sensation, etc., normal. Between 850 and 1200 cubic centimeters of urine were passed daily. The examination gave the following results: Specific gravity, 1014; reac-

tion, acid; color, reddish-yellow; small amount of albumin; no sugar. Microscopically, granular and hyaline casts, red corpuscles, and renal epithelium were discovered.

The patient remained in the hospital thirty-two days, until her death. During this period her temperature varied from 36.7° to 40° C. (98.5° to 104° F.), but for the most part it was regular and continuous. A short time before death it became irregular, and immediately before death there was a rapid decline to 35° C. (95.2° F.).

During the latter part of the illness signs of respiratory and cardiac embarrassment became more pronounced. Dulness, impairment of the breath-sounds, and liquid râles were detected at the bases of the lungs posteriorly; and there was some yellowish expectoration. Gradually the patient grew weaker, the pulse and breathing failed, and death occurred on November 13, 1895.

Autopsy. (Six hours after death.) The body is that of a well-formed young woman. Rigor mortis slight. Postmortem lividity is marked; the ankles are slightly edematous.

On opening the thorax a considerable quantity of clear serous liquid was found in both pleural cavities. There were no adhesions. The lungs were heavy, and on removal both bases were found to be more or less consolidated. This affected the posterior or dependent portions much more than the anterior parts. On section the consolidated areas were found to be mottled in appearance, some areas being slate-colored, others dark red or violaceous. Areas of distinct hemorrhagic infiltration merged insensibly into others which were merely congested. The latter, in turn, were continuous with areas of light color from which liquid flowed abundantly. The upper lobes were somewhat congested and edematous, but not decidedly so.

The pericardium contained but a small quantity of serous liquid. The heart was decidedly enlarged, the hypertrophy involving the left and right ventricles. The superficial vessels seemed normal. A few small "milk-spots" were seen in the epicardium. On section the muscle of the heart was found to be fairly firm, but was rather light-colored. For the most part the muscle seemed granular or opaque, but in places was decidedly fatty in appearance. The cavities of the heart were all somewhat enlarged. A striking peculiarity of the mitral valve became evident at once on opening the left ventricle. Viewed from the ventricular side, this consisted in a pouch-like protrusion from the auricle, lying between the anterior and posterior leaflets of the mitral valve. On close inspection this was found to be an endocardial reflection and had thick chordæ tendineæ attached to it. When examined from the

auricular side, the pouch alluded to was found to be an anomalous appendage of the mitral valve formed in the following manner: At the right extremity of the anterior leaflet of the mitral valve, instead of a free edge, there was an abnormal continuation of the endocardial reflection, attached to the wall of the left auricle and held by thick chordæ tendineæ, springing from a supernumerary papillary muscle in the upper and anterior part of the wall of the left ventricle. The anomalous continuation of the edge of the mitral valve extended from the right border of the anterior leaflet in a serpentine manner upward and then downward upon the wall of the auricle, and was continuous at its other extremity with the right edge of the posterior mitral leaflet. The edge of this adventitious valvular segment, as it may properly be regarded, was contracted and thick, so that the body or part between the attachment and edge was pouch-like, and it was one part of this which projected through the mitral orifice into the ventricle.

In addition to the anomalous valve there was seen in the auricle widespread endocarditis, both old and recent. The wall of the auricle in several places presented small nodular elevations of more or less sclerotic character, and some of them calcareous. The anomalous portion of the valve was thickened in all parts by interstitial change, but in one part was involved by a fresh inflammatory and necrotic process. This extended to the wall of the auricle at the base of the valve, involving this to a considerable distance. The area of disease was in all two or three centimeters in diameter and one centimeter thick, and presented itself as a mass of fibrinous deposit of a friable character, and papillomatous in appearance. Its color was gray or white in most parts, but was somewhat reddish in other portions. When portions of the deposit were torn from their bases the latter were found to be eroded and necrotic in character.

The aortic valves and the valves of the right heart seemed normal in appearance.

Abdomen. The peritoneum was healthy. The stomach and intestines presented no special abnormality. The latter were carefully examined, but beyond some prominence of the lymphoid follicles nothing varying from the normal was discovered. The liver was slightly enlarged, and on section was found to be paler in color and more homogeneous in appearance than normal. The pulp was decidedly friable. The kidneys presented much the same appearance as the liver. They were enlarged and lighter in color than normal; the cortex was wide; the capsules stripped easily. In the left was found a recent hemorrhagic infarction of small size. The spleen was decidedly enlarged and

was very soft. On section, the Malpighian bodies were found to be prominent, and a number of small hemorrhagic infarcts were discovered. The latter were small in size and showed no evidence of retrograde changes. The suprarenal bodies were normal, The pancreas was not noticeably altered. The uterus and its appendages were removed with the bladder and urethra.

On section into the uterus and vagina there was found a considerable amount of mucopurulent exudate in the cervix, and less within the uterus. The mucous membrane was thickened, red, and granular in appearance in the fundus of the uterus, and roughened, but less congested in the cervical region. The vagina contained a certain amount of mucopurulent material, but was not itself noticeably affected. The urethra was somewhat reddened and thickened at the external orifice, but the mucosa throughout the canal was not diseased. The bladder was normal. The Fallopian tubes were both somewhat enlarged, and on section were found to contain slightly turbid liquid. The walls were thicker than normal. The ovaries presented no evidence of disease.

Microscopic and bacteriologic examination. I wish to acknowledge my indebtedness to Dr. S. S. Kneass for his aid in the bacteriologic studies of this case. In the course of our autopsies it had been customary to make cultures from the heart's blood and from the various organs; but as no suspicion had been entertained as to the primary infection in this case we were provided with merely the ordinary agar and serum culture-tubes. Cultures were made from the blood of the right ventricle, from the endocardial vegetations, from the lungs, and from the spleen. Cover-glass preparations were made of the mucopurulent matter found in the uterus, of bits of the vegetations of the heart, and of scrapings from the lungs. (Unfortunately the uterus and its attachments were misplaced, and thus lost.)

Growths were obtained from the lungs, blood of the right ventricle, and spleen. Those from the lungs appeared as a sparse growth upon the surface of the culture-medium, and on microscopic examination proved to be a streptococcus. The blood of the right ventricle and spleen contained a small coccus of the staphylococcus group. The tubes inoculated with bits of the endocardial vegetations remained entirely sterile.

Microscopic examination was made of portions of the various organs (except uterus, etc.) and of the cardiac vegetations. The sections were stained with hematoxylin and eosin, Löffler's blue, Kühne's blue, carbol-thionin, and by Gram's method. The lungs were found to present a mixed form of cellular pneumonia. In many parts the alveoli were filled with large epithelial cells, separated by fluid, or here and there a

leukocyte; in other parts the air spaces were densely packed with leukocytes, while in still others hemorrhagic exudation was pronounced. There were scattered masses of cocci lying in groups like staphylococci and staining with Gram's method or the other methods. No distinct chain-formations or streptococci were discovered. Sections of the liver and kidney showed marked epithelial degeneration, the cells presenting a granular appearance, and the nuclei in many places refusing the stain or staining irregularly. In certain areas complete granular degeneration of the nuclei of the cells was observed. Sections of the spleen were marked by the amount of hemorrhagic infiltration observed. Staining for micro-organisms in these organs was negative. The infarcts were studied with particular care, but no organisms were visible.

The sections and examination of the cardiac vegetations gave surprising results. The scrapings made at the autopsy disclosed diplococci in the leukocytes and to a less extent outside of these. These diplococci were well stained with the blue stains, and especially with thionin; they were decolorized in Gram's stain. The portions, rapidly hardened in alcohol which were sectioned, were found to consist of masses of polynuclear and, to a small extent, mononuclear leukocytes, held together by a small amount of fibrin in the form of a wavy network or of a homogeneous matrix. Stained with Löffler's or Kühne's blue, or especially with thionin, large numbers of diplococci were discovered in and between the leukocytes. Those within the cells invariably occupied the protoplasm, never the nucleus. Those between the cells were very sparse, and were always grouped in pairs, though clusters of these might be associated. In staining the sections it was found that prolonged decolorization with alcohol decolorized the diplococci. This was much more marked in the case of the blue stains and the aniline-oil-gentian violet stain than in the case of thionin. The latter gave by far the most satisfactory results of all the stains employed. It was peculiarly effective when the sections were placed in it for twenty-four hours. The diplococci constantly took on a reddish or violet hue, while the nuclei of the cells were stained a brilliant blue. The nuclei in many parts were almost without exception degenerated and broken into small, round, granulation-like fragments, which made the recognition of the diplococci more difficult in the sections in which other stains were employed. In the sections treated with thionin the micrococci could be readily distinguished from all of such granulations.

In sections treated by Gram's method the organisms were rapidly and completely decolorized. In studying the micrococci more minutely it was found that, though they occurred in pairs almost exclusively, there

were some single forms and other groups in which four were associated in the characteristic tetragonal (merismopedial) form. The separate cocci were in many places characteristically reniform or biscuit shaped, the longer axes being parallel and the concavities opposed to each other. In other parts this appearance did not exist, though in every group of the micro-organisms some at least were of the biscuit-form.

Cover-glass preparations made from the mucopurulent matter in the vagina and cervix uteri disclosed no diplococci, but merely a short bacillus. Unfortunately, the loss of the specimen prevented a more minute examination of the genital organs, and the presence or absence of diplococci in the deeper tissues could not be determined.

In reporting this case as one of probable gonorrheal origin, I am aware that a certain amount of doubt exists, and must therefore detail the reasons that seem to justify its acceptance as a genuine case.

In the first place, the curious condition of the mitral valve requires discussion. This, I have no doubt, was largely a congenital anomaly. The possibility of compensatory hypertrophy of the valvular segments to which Sibson called attention must undoubtedly be admitted in chronic cardiac disease, and I have seen instances which presented such an appearance; but in the present case there was an appendage of such anomalous character and so extensive that the suspicion of a developmental condition could not have been entertained, even if there had not been a distinct supernumerary papillary muscle to evidence the congenital nature of the condition.

Further, there was a distinct history of rheumatism at the age of seven and of cardiac disease at that time. Some of the endocardial lesions also showed that there had been an ancient endocarditis. In the light of these facts the attack of endocarditis which led to the patient's death might be put down as one of "efflorescing" or recurring endocarditis. This, however, does not exclude a new infection; rather, indeed, it favors this idea. My own interpretation of the history is something as follows: Congenital anomaly of mitral valve establishing a place of lesser resistance; rheumatism at seven with acute, terminating in chronic, endocarditis; further loss of resistance at the damaged spot, and finally new infection with fatal endocarditis.

Whether this last infection shall be regarded as gonorrheal or not must depend upon the weight we are disposed to place upon negative evidence. Practically all of the cases of so-called gonorrheal endocarditis reported in the literature have been based upon evidence of the same kind; some, indeed, upon the morphology alone of the organism discovered in the vegetations or elsewhere. At the present time better

proof is required than the mere appearance, size, and grouping of the organisms. While it remains true that the biscuit-shape arrangement and the intracellular position of the organisms are more or less characteristic, these features are not entirely distinctive. Bacteriologists uniformly recognize that staphylococci, streptococci, and certain other organisms may be arranged in pairs in practically identical fashion at some stage of their growth. It is proper, however, to add that this is not regularly the case with any given culture, or in any instance in which such organisms are found in tissues, while the gonococcus habitually, and almost uniformly, presents the grouping alluded to.

The second point of peculiarity that has been relied upon, the intracellular position, is even less satisfactory. A long list of bacteria, including staphylocccci, streptococci, and other micrococci, together with various bacilli, might be prepared to show that this feature is by no means characteristic.

Two forms should be referred to more particularly: the diplococcus intracellularis meningitidis, described by Weichselbaum and Goldschmidt in 1887, and the diplococcus albicans tardissimus, found by Wachholz and Nowak in a medico-legal case of supposed gonorrhea. The former of these two has been studied with greater care, and, while it presents morphologic similarities, is readily distinguished from the gonococcus by the ease with which cultures are obtained on ordinary media, and, according to Jaeger and others, by the fact that decolorization does not occur with Gram's stain when cover-glass preparations are studied, though it does take place in sections. As far as the bearing of these facts upon my own case is concerned, I have no hesitation in excluding this organism. Cultures with the ordinary media failed entirely, though the number of bacteria in the parts of the vegetations implanted upon the media was enormous. Further complete decolorization with Gram's stain was regularly observed.

Allusion has several times been made to the behavior of gonococci toward Gram's stain, and formerly much stress was placed upon this. It must be recognized, however, that other micrococci behave in a similar manner. Bumm described such forms, some of them diplococci, in his work on gonorrheal disease of the mucous membranes, and a number of other authors have reported similarly. Even the staphylococcus in later generations may show this peculiarity, so that the importance of Gram's stain must not be overestimated.

The final diagnostic criterion is the culture. This includes a medium upon which a growth is obtained and the appearance of the growth itself. The difficulty of cultivating the gonococcus is too well known to

require comment. Ordinary media, such as agar, glycerin-agar, or the ordinary blood-serum, remain sterile. This fact is one of great importance in the diagnosis of some of the reported cases in which no culture of any kind could be obtained, and it was particularly important in my own case. The ease with which I have obtained growths from other cases of endocarditis (due to staphylococci and streptococci) contrasts strongly with their entire absence in this case, and especially as the microscopic examination showed such a wealth of micrococci. As far as our knowledge of bacteria extends, the gonococcus is the only organism of similar morphology likely to behave in this manner.

Taking the evidence all in all, I am therefore led to believe that this case was one of true gonorrheal endocarditis, and not one of a second or other infection. The facts favoring this view are briefly: the entire correspondence with gonococci in morphology, position, and behavior to stains; and the entire failure of cultivation-experiments.

November 14, 1895.

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7. *Report of a case of abdominal aneurysm.*

Exhibited by WILLIAM W. YOUNG, M.D.

Hugh R., white; aged forty-nine years; born and residing in Philadelphia; admitted to Philadelphia Hospital August 24, 1895.

Family History. Parents dead: father of some form of dropsy; mother, after a long illness, in which jaundice was a prominent symptom; one sister died of dropsy, which began in the feet and gradually ascended; one in confinement; one brother died after an operation for removal of a tumor of head of femur caused by traumatism. After the patient's death it transpired that he had a wife and two seemingly healthy children in Trenton, N. J., and that the wife had been treated in this hospital some years previously for syphilis, contracted from the husband. Whether before or after birth of the children is not known to a certainty.

Previous History. Has often been sick; frequently from rheumatism, so-called, and lost sleep at night by reason of pain; has had pain in the back for some years, and referred it always to kidneys being affected; had scarlet fever at age of nineteen years; gonorrhea several times, and stricture as a result; treated at this hospital; primary syphilitic lesion nineteen years ago; malaria in 1877, and in childhood some minor

affections; grippe two successive years; lately often felt dull and listless; never sick enough to keep him from work; always drank heavily and continuously, and used tobacco freely, both chewing and smoking.

Present Trouble. This history was taken October 21, 1895. Three months ago began to have pain in side and vomiting-spells; lassitude; loss of sleep and appetite, and felt unable to work. Admitted to this house with these symptoms and a dull, numb feeling down the leg. After admission noticed a pulsation affecting the left hypochondrium; slow, expansile heaving, developing gradually and extending to the median line, anteriorly and posteriorly, most noticeable to the left and posteriorly about two inches below costal margin and in the post-axillary line. At this point bruit is heard with difficulty by immediate auscultation and only with the subsiding wave of the pulsation. This condition existed for some time, the prominent symptoms being pain in back, increased by motion and upright posture, loss of sleep, vomiting, weakness, shooting pain from back to left thigh and knee, once shifting to right side, to return again to left.

Cough of no great severity and with little expectoration; some constipation, the overcoming of which did not lessen pulsation; urine normal in specific gravity and constituents, and varying from fifteen to twenty-eight ounces daily; appetite poor, vomiting a variable symptom, no pupillary changes; reflexes generally good; pain increased by pressure over the pulsating area always; the extent of the tumor in the hypochondrium was doubtful, as it was overlain by the intestine and extended upward to within the costal margin, but no signs could be detected to the right of median line, either anteriorly or posteriorly, and "bruit" was heard only at the single spot previously designated. Pulse was full, strong, slow, and regular.

October 25, 1895. After observations, extending over two months from time of admission, Dr. William E. Hughes and Dr. S. Solis-Cohen have agreed that there is here present an aneurysm of the abdominal aorta, this diagnosis being also made independently by Dr. John Deaver, who refused to interfere surgically, saying that there was no possibility of its being anything but an affection of the main trunk, and, therefore, operation would be useless. During the time previous to this the symptoms were sometimes so slight as to render the diagnosis doubtful.

On examination to-day the pulsation is changed in character and position; now it is situated well up under the left thorax and extends past the median line anteriorly. The stomach seems less encroached upon. Pulse is quicker and more rapid, and, therefore, the aneurysmal

pulsation is less and not so painful; bruit is now plainly heard, plainest in epigastrium; chest is heaving at the rate of 18 per minute; there are no shooting pains, merely a numb feeling over affected area; no vomiting.

November 2, 1895. *Heart.* There has always been a mitral systolic murmur; the position of dulness and the impulse changes from day to day; position of impulse tends to travel upward, and is now at fifth to seventh ribs, left side, and more jerky and frequent than formerly.

The *double bruit* is seemingly close to the ear in the normal area of cardiac dulness, but the sound cannot be separated so as to differentiate whether the phenomenon be due to the aneurysm or the contraction of the heart itself.

The chest-measurement on the left side at base is nineteen inches, while on the right side seventeen and one-eighth inches is the measurement.

Expansion. Left, *nil;* right, one-quarter of an inch.

Dulness has slowly grown up along the midsternal line to third intercostal space, extending gradually about the chest under the axilla to the spine, and from xiphoid cartilage to one inch below the costal margin, following this in a parallel manner to left hypochondrium, midaxillary line, and dipping down and backward to the lumbosacral junction, there merging into the dulness of the spine and gluteal mass. There are no spots of resonance in the back on the left side, and examination over this area causes pain to be experienced for some hours afterward. The impulse for four or five days has been stationary—as here described—and most marked at midclavicle between fifth and seventh ribs (November 2, 1895). At third interspace, midclavicular line, a bubbling sound is heard both by the unaided ear and stethoscope, synchronous with the pause between inspiration and expiration. Tubular breathing present. Above third interspace on left percussion gives Skoda's resonance. Below this point, with tolerable regularity along the interspace as a dividing line, is found dulness. Palpation shows loss of tactile fremitus below the line mentioned and its presence above slightly less than normal (probably due to the lessening of entire lung area doing functional service). Egophony also present.

Right lung shows compression by a high, sharp note on percussion, and dulness extends up to the fourth interspace, becoming lost below in that of the liver. The subjective symptoms are now mostly dyspnea and pain, with a sharp, shooting pain in the left scapular region.

November 5, 1895. Dulness to-day extends on left side to third interspace, from midsternum to spinal column, following the interspace irregularly. In the area below this line, following the old line of demar-

cation, below, breath-sounds are absent, as are voice-sounds and fremitus. Above the line the sounds and signs previously noted are present in a still more exaggerated condition, viz., egophony, increased fremitus, increased high-pitched percussion-resonance with an almost metallic quality, and bubbling râles, large, and heard close to the ear. On the right the change is not at all marked, and is only noticeable in the still more embarrassed breathing. No heart-sounds are discernible as such, only a confused jumble of sounds, with a double time in a general way, but mixed with the gurgle of the fluid present in the pleural cavities and heard as rather distant sounds.

The pulse is markedly weak and dicrotic, the dicrotic wave being almost as strong as the actual impulse. Pain is intense, most marked on motion, and situated in the back. Once, on assuming the sitting posture, he complained of paralysis in the left leg, and the assistant had to lift him into bed, the leg being quite useless. After this he could not raise his body from the bed, and the enlargement of the left chest seemed more pronounced. Attempts to pass the tapes gave so much pain that they were not persisted in, and no measurement was made.

Facial expression at this time was very instructive as indicating exquisite pain. Delirium was present, and the patient seemed quite anxious, as though fearing some serious occurrence imminent. The delirium was of low type.

Urine, eighteen ounces and less in twenty four hours, albuminous, and containing numerous granular casts. Stools only voided with difficulty after the desire for defecation is obtained by drugs. Cyanosis present about face and finger-tips.

On evening rounds to-day, November 5, 1895, the patient, with a characteristic stubbornness, said he would sit up and eat his breakfast in the morning. This was 7.30 P.M., at 9.30 P.M. he was dead, presenting the appearances attendant upon internal hemorrhage and looking more as though he had been dead ten hours than ten minutes.

Now the dulness seemed somewhat less than during life, but returned after twelve hours as before, so that at postmortem examination all signs on percussion were as noted during life.

Postmortem. By Dr. E. B. Sangree. Dictation by Dr. W. E. Hughes. Body well nourished. Rigor mortis well marked.

Abdominal cavity contains small black clots of semifluid blood. Stomach lies almost vertically, its left border corresponding to the middle line.

Liver lies also almost vertically, but less so than the stomach, its left lobe being pushed up and its right lobe downward. Diaphragm is displaced to fourth interspace on right side.

The whole of the left half of the abdominal cavity to within two inches of the ilium is filled out with a moderately firm mass, carrying in front of it the colon, which is dissected off from it with difficulty. The anterior wall of the mass measures in length thirty, breadth eighteen, depth ten centimeters. It is so adherent laterally and posteriorly that it can be taken out only by dissection.

It is composed centrally of firm, laminated clot, peripherally of soft, black clot merging into the laminated clot.

The rupture into the abdominal cavity has apparently taken place posteriorly and a little also at the lower border of the spleen. The aneurysmal cavity communicates with the aorta by an opening in the posterior wall of the aorta seven centimeters in length, involving about one-fourth of the circumference of the vessel, the lower edge of the opening being opposite the inferior mesenteric artery. This opening has firm, round, and smooth edges directly continuous with the lining of the aneurysmal sac.

Left pleural cavity contains about a half pint of clear, serous fluid, covering the lung. Pressure upon the lung causes this fluid to become blood stained.

Right pleural cavity contains also a quantity of serous effusion. There are no parietal adhesions in either cavity, but the left base is strongly bound down to the diaphragmatic pleura.

Pericardial sac contains small quantity of clear fluid; no adhesions. It and the heart, in common with other organs, are displaced upward and to the right. Heart contains some fibrous clot. Mitral valve a little thickened. Muscle quite healthy.

Left lung completely atelectatic in lower lobe and lower half of upper lobe. Upper lobe normal in appearance of upper half, and the line between the two areas well marked. Lower lobe adherent to diaphragm; adhesions fairly well organized. Left lung compressed; lower lobe entirely flat and extending to third interspace. Right lung effuses a large quantity of fluid on pressure, clear in upper lobe, blood-stained in lower. Upper and middle lobes strongly adherent. Adhesions edematous. Right lung pushed by intra-abdominal pressure up to the fourth rib.

Spleen constitutes part of the upper and anterior capsule of aneurysm. Length, 10.5 centimeters; breadth, 9 centimeters. It is flattened, substance superficially soft and strikingly pale, and toward the posterior extremity pale-yellow and slightly fibrous. The portion lying beneath the softened and yellow part is eroded by the aneurysm without infiltrating its substance. The left suprarenal is very friable, light-

yellow in color, and embedded in a clot of blood immediately beneath the aneurysmal sac.

Left kidney constitutes in part the wall of the aneurysm and a part is embedded in clot. It is flattened, resembling normal spleen (on section). In the upper part no differentiation between cortex and medulla can be made. As the lower part is approached, the texture is more normal, and differentiation can be made, but the cortex is diminished, is very homogeneous and shiny. In the lower end irregular areas of cortex are light-yellow, and suggest purulent infiltration. Pelvis little dilated; filled with a purulent, blood-stained liquid. Vessels patulous: organ not weighed, as it would destroy specimen to dissect it away. Right suprarenal, normal.

Right Kidney. Cortex noticeably increased. Surface pale and quite irregular. On section surface is translucent and shiny, and is uniformly pale. Weight, 270 grams. Except in location, the stomach and duodenum are normal.

Pancreas forms part of the wall of the aneurysm, and is rather firm.

Liver firm, surface mottled by pallor of the periphery of the lobules. The left extremity of the left lobe is atrophied by pressure of the growth. There is a well-marked line between the atrophic and non-atrophic portions.

Aorta. Thoracic aorta somewhat dilated. Few atheromatous patches. Abdominal aorta quite atheromatous, especially in the region of the rupture.

Bodies of the twelfth dorsal and first lumbar vertebræ extensively eroded. Second lumbar vertebra less so. Lower ribs also eroded, the last one entirely cut through on the left side. *December* 12, 1895.

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8. *Old pericarditis; mitral and aortic obstruction.*

Exhibited by AUGUSTUS A. ESHNER, M.D.

I was hastily summoned, early one morning, to see F. B., thirty-three years old, a waiter, with the information that the man was dying, but upon my arrival found that he was already dead. The man had consulted Dr. J. C. Wilson on three occasions, and the existence of cardiac valvular disease, namely, mitral stenosis with dilatation, and failing heart-muscle had been recognized. He had latterly complained most of symptoms of gastric disturbances, and had obtained subjective relief from ten

drops of tincture of digitalis taken four times a day. The man had been at work on both the day and night before his death, and had retired to bed at about ten o'clock, after smoking a pipe, with the intention of arising early on the following morning. In the course of the night he got up and went into the bath-room nearby. Later his wife heard him gasp or groan, and, failing to receive an answer to her calls, went to his aid, but without avail. I subsequently learned that the man had had an attack of acute articular rheumatism, four years ago, that kept him in bed for about two months, and in his room for an additional month and more. In the course of this illness he was on one occasion seized with syncope, and would probably have fallen had his wife not supported him. Two years later he had a subacute rheumatic attack. Since these attacks the man had at different times suffered with precordial pain, dyspnea, palpitation of the heart, vertigo, headache, and cyanosis of the lips and nails.

Upon postmortem examination the pericardial cavity was found obliterated by well-organized but yielding universal adhesions. The greatly enlarged and dilated heart was distended with dark blood, largely fluid, and its muscular walls thickened. The leaflets of the mitral valve were thickened and bound down, and the auriculoventricular foramen was contracted so as to admit only an index-finger. The chordæ tendineæ as well as the papillary muscles, in part, were thickened and contracted. The aortic semilunar leaflets were likewise thickened and deformed, and restricted in mobility. The specimen is a typical one of old pericarditis and endocarditis of the left side of the heart, with their structural sequelæ. *December* 26, 1895.

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9. *Fibroid heart and cystic kidney.*

Exhibited by H. A. Hare, M.D.

R. L., sixty-five years old, married, German.

Family history. Father died of apoplexy, two years ago. Mother died of dropsy.

Personal history. Patient of good habits, no alcoholic nor specific history. Suffered with the usual diseases of childhood, otherwise well until two years ago, when he had an attack of rheumatism. Since then has had cardiac symptoms in a slight degree. Two months ago these symptoms suddenly became pronounced, and upon ascending stairs he had a violent attack of dyspnea, and palpitation of the heart. A

few days later the symptoms became still more aggravated, and he noticed marked swelling in his feet. A dry cough also developed, and five days previous to his admission he was unconscious for fifteen minutes. His urine has lately become diminished in quantity. Appetite fair, bowels regular. Temperature 98° F., pulse 106, respiration 30; patient markedly cyanotic, pulse weak, heart-sounds feeble. Had an arcus senilis most marked in upper segment of corneoscleral junction. Had a violent attack of dyspnea shortly after admission. Suffered no pain. The urine contained albumin.

Postmortem notes. Body well-nourished and very muscular; slight edema about the ankles; rigor mortis not yet developed; slight postmortem lividity over posterior aspect of trunk; the right cornea is slightly opaque; the subcutaneous fat is present in fair proportions, and the muscular tissue is dark-red in color, and healthy in appearance.

The abdominal cavity. The omentum and mesentery are loaded with fat, and the veins are greatly distended. In the cavity there is about one quart of clear serous fluid. The gall-bladder protrudes one inch beyond the anterior margin of the liver. The anterior margin of the liver is even with the margin of the costal cartilages.

Pleural sacs. The left pleural sac contains one quart of clear serous fluid. There are several slight fibrinous adhesions at the apex of the lung, and a large fibrous adhesion at the side of the lower lobe, near the base. The right pleural sac contains three quarts of clear serous fluid. There are a number of delicate fibrinous adhesions at the apex of the lung.

Pericardium. The membrane appears normal, though the sac contains four fluidounces of clear, serous fluid.

Heart. All the walls of the heart are relaxed; the right auricle is greatly dilated, and contains large mixed clots and a small amount of fluid blood. The right ventricle is dilated, and contains large currant-jelly clots, fluid blood, and tough antemortem clots. The left auricle is slightly dilated and filled with currant-jelly clots and fluid blood. The left ventricle is greatly dilated, its capacity being fully ten fluidounces; it contains fluid blood, and large currant-jelly clots. The tricuspid valves are thickened and slightly contracted; the orifice is slightly dilated. The pulmonary valves are competent. The mitral valves are opaque and thickened, especially the anterior leaflet. In the centre of the anterior leaflet are several small, hard nodules. The aortic valves are competent, although they are thickened and somewhat rigid. On the aortic side of the leaflets are many small points of calcareous infiltration. The wall of the left ventricle is much thinner than normal. The muscular tissue of the septum and external wall along the right side of the heart is

the seat of marked fibroid changes. This tissue cuts with greatly increased resistance, and running through it may be seen fine fibrous bands. The endocardium covering this area is white in color, opaque, and greatly thickened. The endocardium covering many of the columnæ carneæ, and the musculæ papillares, is thickened and the muscular tissue beneath fibroid. The aorta and coronary arteries are atheromatous.

Left lung. There are several small calcareous nodules about the size of a pea in the apex. On section the surface is dry and grayish in color. The bronchial tubes are the seat of a chronic catarrhal inflammation. *Right lung.* The right lung is in about the same condition as the left.

Spleen. There are a number of delicate adhesions about the spleen. The capsule is tense, and at places greatly thickened, white, and opaque. At the back of the spleen a round nodule projects slightly above the surface. On section the organ is very dark in color and quite firm. The nodule previously noted is an old infarction area, and it extends almost completely through the spleen.

Left kidney. The lower half has undergone cystic degeneration; the kidney-structure in the upper half is of a very dark-red color and quite tense. *Right kidney.* The right kidney is slightly larger than normal, and dark-red in color. On section it cuts with marked resistance. The surface line of the cortex is slightly irregular, though over the greater number of pyramids it is of normal thickness. The cortex and pyramids are dark-red in color, with yellow striations. The tips of the pyramids are yellow. The capsule strips off readily and exposes a smooth, shining surface.

Liver. The liver is very small and much lighter in color than the normal; the surface is granular and irregular; it cuts with marked resistance; the portal vein branches are distended and filled with fluid blood.

Stomach and duodenum and small intestines. The veins of these organs are distended and engorged with blood. The organs above named are affected also by a chronic catarrhal inflammation. *February* 27, 1896.

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10. *Dissecting aneurysm of the aorta.*

Exhibited by Herbert U. Williams, M.D.

The account of the clinical aspect of this case I owe to the courtesy of Dr. Charles G. Stockton, who attended the patient.

On the 27th of September, 1894, I examined George R., fifty-eight

years old, and of German parentage; occupation, hotel-keeper. His father died at the age of seventy-three years; his mother, four brothers, and one sister are living and well. One sister died at the age of forty-three years of pericarditis, with effusion, following rheumatism.

Patient was never sick until eight years ago, when he had pneumonia, from which he suffered a severe relapse. Last winter (about ten months since) he suffered from shortness of breath, with heavy beating of the heart, as he called it, "pounding of the chest," accompanied by pain and a sense of soreness when walking in the cold air. Since that time he has been short-winded on exercise or excitement. The patient was five feet five inches high, and, at the time of his examination, weighed, stripped, 216 pounds.

There was a decided lividity in his face, with blueness of the lips and tongue. His pulse was weak and irregular; his capillary circulation was poor. He had a dry, asthmatic cough, with very scant mucous expectoration. His appetite and digestion were good; his bowels regular.

The physical examination revealed emphysema in both lungs, with congestion at the bases. Examination of the heart showed a systolic apical bruit heard far to the left of the nipple-line. The impulse was diffused and almost indistinguishable. The pulmonary second sound was over-accentuated when the patient was in the upright position; and, when lying down, the patient's face became congested; then the aortic second sound was much more marked than the pulmonary. Owing to the emphysema, it was not easy to describe exactly the left border of the heart.

He voided 1824 c.c. of urine in twenty-four hours, which contained 17.888 grams of urea. The specific gravity was 1010, and it contained a trace of albumin, but no sugar nor indican. Microscopically, there were found triple phosphate crystals.

Under treatment the patient improved considerably in his symptoms and continued with his business, in spite of objections raised by his physician and friends. He was in the habit of walking about the streets, feeling well save for his shortness of breath. One month before coming to me he had passed an examination for life-insurance. He was carefully examined for aneurysm, but no physical signs of such a condition could be discovered. He was found dead in his bed, without having made any complaint in the night.

Autopsy, at the patient's residence, November 1, 1894, 4 P.M., about twelve hours after death. Body cold; rigor mortis firm; usual post-mortem discoloration of dependent parts; very fat man; subcutaneous

fat three-quarters inch thick over thorax, two inches over abdomen, and abundant subperitoneal fat; costal cartilages calcified; opening left pleural cavity disclosed a quantity of bloody serum and an enormous, firm, dark blood clot; the left pleura was found to be forced downward, having been dissected from the chest-wall in part, making a cavity above and behind the lung, full of clotted blood, and communicating with the pleural cavity; right pleural cavity empty; lungs crepitate well and seem normal; heart very large, abundant supercardial fat, two ecchymotic spots on anterior aspect; muscle thick, firm; hypertrophy and dilatation, especially of left ventricle; mitral orifice admitted two fingers; segments of all valves attenuated and stretched, but not thickened or giving evidences of endocarditis. Moderate atheroma of ascending aorta.

Leaving the aorta for the present to relate the condition of the other organs briefly: Left kidney large, firm; external surface showed a number of small cysts, and one as large as a hickory-nut; capsule somewhat adherent; surface beneath it granular; cortex narrow, with alternate pale and red striæ.

Right kidney similar to the left.

Histologic examination subsequently showed the changes of chronic diffuse nephritis.

Spleen and liver not remarkable; gall-bladder contained about one-half ounce of bile and a good-sized gallstone. Stomach small, and large intestines and vermiform appendix normal. Pancreas and central nervous system not examined.

To proceed with the examination of the aorta, its upper and posterior wall close to the left subclavian artery exhibited an opening one-quarter inch in diameter, approximately round, which at first I supposed to communicate with a dissecting aneurysm encircling the aorta, rupturing about the left lung and finally into its pleural cavity. Such an aneurysm certainly existed, and its rupture was the cause of death. The aneurysmal dissection in the thoracic aorta appeared to the left both in front and behind, crossing anteriorly to the right and behind, coming in the lower thoracic and upper abdominal aorta to nearly encircle the entire trunk, and finally to do so completely, reaching as low as the bifurcation, and down the left common iliac, one and one-half inches on the internal and two inches on the external iliac, while it passed down the right common iliac to its bifurcation.

I have seen another dissecting aneurysm of the aorta much like this one, but of even greater extent.

I discovered that the opening described as lying close to the left sub-

clavian artery communicated, not with the aneurysm directly, but with a vessel running parallel with the aorta and to the left of it, and that a probe inserted into this vessel would enter the left external or internal iliac arteries. A probe passed into the aorta entered the right common iliac.

The dissecting aneurysm surrounded not one but both vessels, more or less completely, lying between the middle and outer coats, or between the outer coats and the sheath. It had its origin in a rupture in the wall, not of the main aorta, but of the channel lying to the left of it. Breaking through its thin covering, it had stripped the pleura from the left thoracic wall. Finally, it broke through the pleura also; hence, the extensive hemorrhage into the pleural cavity.

Of the two vessels, the right is the larger, and is in a direct line with the descending limb of the arch. The right vessel exhibits marked arteriosclerotic changes in the thoracic region. The small and rather ragged opening of the left vessel into the right looks more like a pathologic than a natural outlet. The left vessel does not exhibit arteriosclerosis.

The principal arteries arise from the right branch, except the inferior mesenteric, which is given off from the left, behind. A peculiar origin is that of a trunk which I suppose to be the left renal; it leaves the right vessel close to the septum, between that and the left; while the left side contributes to its origin in part, and exhibits an opening apparently destined to enter the same vessel, but which ends blindly. The other large trunks, though not at all typical in their arrangement, are not strikingly abnormal.

There are ten pairs of intercostal arteries arranged along the right vessel, mostly patulous, but a few of them not so, and some of them indicated only by depressions. The closure, in one or two cases at least, may have been effected by arteriosclerosis.

I can only detect the first two pairs of lumbars; but the others will probably come into view when the specimen is opened lower down than has yet been done.

A very remarkable feature is that many of these small arteries open from the right vessel into the left, and go no farther. That is true for ten little arteries from the eighth intercostal to the second lumbar, inclusive. The left vessel apparently carried out the functions of these arteries by branches of its own, of which there are nine in all, at corresponding levels. The exact arrangement of the branches from the respective vessels will only be clear when both have been split open from end to end. I have preferred first to exhibit the specimen as it is,

finding that as dissection proceeded the peculiar features became obscured.

Histology of aorta. The right-hand vessel is, aside from arteriosclerotic changes, of normal structure. The left is extremely atypical, showing relatively little elastic tissue, but a dense, lamellar, fibrous, connective tissue, with many nuclei. The peculiarities of each half are transmitted to the common septum. A sheath belonging to both vessels surrounds them as they lie side by side.

[NOTE.—The writer furthermore expressed the opinion that the condition was best explained as a defect of development, originating in the duplicate condition of the aorta present in the embryo at an early period. A positive decision could only be reached after the completion of the dissection, which had been deferred to permit of the exhibition of the specimen at this meeting.

When the parallel vessels were opened subsequently, the right and larger one was found to bifurcate, after the manner of the normal aorta, into two common iliacs, although a probe passed into it from above had entered only the right common iliac. The left of the two large parallel vessels followed the course of the left common iliac, and ended by dividing into two portions, one of which opened into the left internal and the other into the left external iliac.]

DISCUSSION.—DR. ALFRED STENGEL had a specimen very much like this, which was presented to the Society about a year ago by Dr. Cattell, and which was taken from a man who had lived to about forty-eight years of age. He did not present any symptoms at all of a vascular character. He was admitted to the University Hospital, suffering from an affection which was supposed to be due to syphilis, which he admitted having had some years before. As far as the heart and arterial system were concerned, no symptoms were made out. He was placed on antisyphilitic treatment and given inunctions. He became salivated after three inunctions of a dram each of mercuric ointment, passed into coma and died, and the cause was never known. A post-mortem was made, and the only striking thing was a large dissecting aneurysm, which began in about the same situation as the upper opening in the present case. It extended the entire length of the aorta, and opened again into the iliac branches and into the root of the aorta just before its bifurcation. The aorta was converted into a double tube, resembling the present specimen very closely. These tubes on examination were found to be composed of the coats of the aorta, dissected

and changed by the process of disease, and the structure could not be made out. The adventitious coat was lined, as seems to be the case in this specimen, by thickened plates of fibrous tissue, and in some places it had undergone secondary calcification. The appearance was that of atheromatous plates as they occur in normally lined bloodvessels. He would refer to the discoveries made in cases of this kind, particularly to those of Peacock, who made an elaborate study of dissecting aneurysms. He found that they were sometimes lined with endothelium, and he explained it as being a growth from the point of rupture downward, through the aneurysm. One could readily understand how an error of observation might be made in these cases. The lining may be true endothelium, and this was Peacock's opinion. In the case I refer to there was the peculiarity that was present here, and this, it seems to me, makes it somewhat doubtful whether it is really a double aorta or an aneurysmal dilatation. There were two other reasons why he was rather dubious as to its being a double aorta, whether congenital or not. If it were due to the improper union of an embryonic double aorta, we should scarcely expect such an opening as existed here. Again, if this were a division, the appearance would be quite different. This had the appearance of ruptures into dissecting aneurysms, as we usually found them. Another thing which made it questionable was the histologic structure. Dissecting aneurysms were found dissecting between the coats of the media, and the muscle-coats are drawn to the other side. In such a case, if the division came near to the outer limits of the muscularis, then the external limiting elastic membrane would be carried outward. If the muscular coat were penetrated or dissected near to its inner limit, then the internal elastic membrane would be carried to that side. The muscle-tissue would be apt to be more or less transformed, so that its real character would be changed. On one side would be a strong, fibrous tissue, with, perhaps, fibro-elastic tissue, and very little muscle, or some muscle transformed into the appearance of fibrous tissue, and on the other side the elastic tissue of the intima more or less thickened. The appearance, as he understood from Dr. Williams' description, was that the right branch had the normal coats, but less fibrous tissue and less elastic tissue than the other, so that it would seem as if there had been a division in the outer muscularis, between it and the external elastic tissue. That would also explain the description of one of the branches to which Dr. Williams refers. It seemed to him that this was the strongest proof of all that this was possibly an aneurysm. If it was an aneurysm, then there was a second one. A case of this kind was unique. Where the intima and the media have

broken and allowed aneurysmal dilatation, there is apt to be a rupture of the remaining coats of the media and parts of the adventitious tissue. If this was really a double aorta, it was a most unusual and interesting specimen. It certainly had appearances which made the question difficult to decide, but he inclined to the other view.

Dr. Eshner said he hesitated to speak on this matter, as he was not sufficiently familiar with the process of development. His impression was that when the heart divided into its four chambers the arrangement of the vessels was rather symmetric, and that the two aortas arose somewhat in the way he would show by a diagram. The point of difficulty to be explained was the arrangement of the division. It was not really a division. One part formed the left iliac and the other the right. That seemed likely to him to be two distinct vessels.

Dr. F. A. Packard thought that it would be only proper to speak of the specimen as two aortas if there were distinct histologic elements. If it were simply an aorta divided by a septum the same histologic elements should be present in both. Looking at it casually, it looked to him like a double aneurysm with a secondary aneurysm. The same point occurred to him that Dr. Stengel had mentioned: If it were a double aorta the point of entrance would have to be decidedly larger, but this did not prove the idea, however. The second aorta might receive blood from the communicating branches. The orifice into the second aorta was extremely small.

Dr. W. E. Hughes inclined to the views expressed by Drs. Stengel and Packard, and thought it was an example of a very curious dissecting aneurysm. The apparently complete absence of the intima made this exceedingly probable. The fact that there was arteriosclerosis, which was a disease of the intima, in one branch would argue for this. It was just as easy for an aneurysm to break about the left common iliac as it was for it to break about the renal. He believed there were examples on record in which a dissecting aneurysm had communicated with one iliac and the normal aorta with the other. As far as the opening went, it might be a normal opening into the second aorta. It need not be any larger than that, considering that there was a great deal of arteriosclerosis which might possibly be conceded to occlude the original opening. The fact that the sclerosis did not extend down through the opening was a proof that the second trunk was a dissecting aneurysm.

Dr. Williams, in closing, said his object in bringing the specimen to Philadelphia was to get just the kind of a reception which it had gotten, and he was very glad that he had brought it. He was not entirely convinced in the matter, as he had gone over its appearance

thoroughly. There was only one way of settling it. The size of the opening attracted attention. It seemed insufficient to allow blood to enter into the entire left iliac. There was a quantity of communicating branches, however, which would have been able to help out without difficulty. The raggedness of the opening was suspicious, although, as Dr. Hughes had said, the arteriosclerosis would help account for that. The absence of sclerosis on the left side would seem not to be an objection, as it might be, because the tension was less on that side. It would be a pretty illustration of the pathology of this subject. The existence of branches on the left side corresponding to vessels on the right side was suspicious that the intercostals might have been turned away and the communication established in that manner. The two vessels were symmetric, however. On the histology of the vessel he should not be disposed to put as much weight as had been done. The abnormal vessel might have strong abnormalities in its structure, and did not need to be exactly like the normal. It might show embryonic elements in its structure. These might undergo various kinds of metamorphoses. It seemed incomprehensible that complete communication should have been established with a dissecting aneurysm and the left iliac vessel, with no evidence of the iliac having communicated with the main aorta. If the septum continued complete all the way down, and the left half coincided with the left aorta and the right with the right, we should hold to his theory. If one could find some communication which he had not found he should be compelled to alter his view.

The PRESIDENT said the Society would be very glad indeed to get a further report, and the members would be pleased to have the history of the case for the TRANSACTIONS, if Dr. Williams would be kind enough to supply it. *February* 27, 1896.

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11. *The heart and other specimens from a case of pernicious anemia.*

Exhibited by A. E. TAYLOR, M.D.

The patient from whom these specimens were taken was a man, aged thirty years, who died recently in, what I beg permission to term, his third attack of pernicious anemia. Three years ago he had his first attack, in which his red corpuscles fell to 1,000,000 in the c.mm.; under the use of arsenic he recovered. A year ago he relapsed, and was treated in the Howard Hospital by Dr. Stengel. Although his red

corpuscles fell to 600,000, under the use of arsenic and bone-marrow he again regained fair health. This winter he entered the University Hospital very anemic and very sick. Aside from the common symptoms of this form of anemia, he presented more unusual signs. The heart was very considerably enlarged, and presented a strong systolic mitral murmur of doubtful character. Persistent vomiting was another prominent symptom. Despite arsenic and bone-marrow he sank steadily and died.

On admission a blood-count gave the following result: hemoglobin, 30 per cent.; red corpuscles, 950,000; leukocytes, 12,000. There was only moderate poikilocytosis, and but few nucleated red cells, yet of these few several were gigantoblasts whose nuclei presented karyokinetic figures. The resistance of the red cells was estimated by Hamburger's method, and determined to be very appreciably reduced. A six-tenths per cent. NaCl solution is currently spoken of as a "normal salt-solution." In truth such a solution is normal only for the frog, from whose blood the determinations were originally made. A salt-solution "normal" for healthy human blood, that is, a saline solution of such concentration that it is isotonic to the red corpuscles, is a four-tenths per cent. NaCl solution. In this case so reduced was the resistance of the red cells that a saline solution of between six-tenths and seven-tenths per cent. was necessary to preserve the hemoglobin unaltered in the stroma of the red cells. A differential count of the leukocytes gave the following result:

Neutrophilic polynuclear leukocytes	64.0 per cent.
Eosinophilic polynuclear leukocytes	1.5 " "
Simple mononuclear leukocytes	8.0 " "
Eosinophilic mononuclear leukocytes	0.5 " "
Simple transitional leukocytes	4.0 " "
Eosinophilic transitional leukocytes	0.5 " "
Lymphocytes	22.5 " "

At no time was I able to find any myelocytes or karyokinetic figures, or siderosis in the leukocytes contained in finger-blood. Siderosis was, however, seen in a very few cells from the heart's blood. As the disease progressed the percentage of mononuclear leukocytes steadily increased, while the percentage of polynuclear cells decreased. Just before death the differential count of leukocytes showed the percentage of polynuclear elements had fallen to 50 per cent., while the number of large mononuclear cells and lymphocytes had risen to 12 per cent. and 30 per cent. respectively. This condition is very interesting. Ehrlich looked upon it simply as an index or result of blood-debility. Because the view that the red cells develop from the lymphocytes has been re-

jected, it has been impossible to regard the condition as a sign of attempt at blood-regeneration. In the February number of Virchow's *Archiv* (1896), however, Neumann returns to the older view as a result of years of study on frog's blood: He believes to have observed in frog's blood all stages of gradual development, from the simple lymphocytes to the "spindle-cell," and from this cell to the red corpuscle. Should these observations be confirmed it is probable that some definite meaning can be attached to the excess of mononuclear elements. From another stand-point the condition may be regarded as a result of some intoxication.

The count of the blood-plaques varied between 100,000 and 500,000 per c.mm. The blood presented distinct hyperinosis; the blood clotted instantly, and the clot contracted normally. The specific gravity of the blood (determined by a 6-gram picnometer) was 1029, fully twenty points too low. The alkalinity corresponded to 140 mm. NaOH in 100 c.c. of blood, which means little after all allowances are made for the obvious inaccuracy of the method employed (titrations with oxalic acid solutions). There was a trace of hemoglobinuria and albumosuria. No excess of xanthin-bases, and no lactic acid.

As the man became worse his blood-count became lower until before death it was: hemoglobin, 22 per cent.; red cells, 650,000; white, 6000. I desire to call particular attention to the fact that at all times his hemoglobin-percentage was higher than his percentage of red corpuscles. I wish also to call attention to the very moderate irregularity of form presented by the red cells in this case. I have here six specimens of the blood of as many different cases of pernicious anemia, and one slide from a case of chlorosis; in only one of the slides of pernicious anemia blood is the poikilocytosis marked, and most marked of all is the irregularity of form presented by the cells of the chlorotic blood. The urine of this patient at no time gave any reaction of albumin to the clinical tests; no sugar; at times it contained a few hyaline casts.

At the postmortem examination the body, very yellow in color, was not at all emaciated, rather the contrary, quite fat, the subcutaneous and omental fat being of a deep orange color. No hemorrhages were anywhere found. All the tissues presented fatty degeneration; all the tissues showed on microscopic section fatty degeneration of the essential cells, and a marked proliferation of connective tissues; in addition, scattered everywhere throughout the body were myriads of basophilic cells, the "mastzellen" of the Germans, round or oval cells measuring 12 to 25 μ, with a palely-stained nucleus; the protoplasm full of granules, which stain intensely with the basic aniline stains. In only one

tissue, the suprarenal capsules, were these cells wanting. In addition the different tissues presented individual changes.

The heart, which I have here, is very large—hypertrophy with marked dilatation; without valvular lesions, but with the valves insufficient for the dilated cavities. The substance is fibrous, as well as fatty, and shows no abnormal pigment. The lungs were edematous. The mucous membrane of the stomach was quite smooth, and presented, beside a moderate degeneration of the glandular tissue, a very marked fibrous proliferation, a condition likewise seen in the duodenum. The liver was very fatty, friable, and contained an excess of iron-pigment deposited in the periportal connective tissue and perivascular and peribiliary lymphatic tissue. At no place have I been able to find iron-pigment in a liver-cell. The spleen was enlarged, very hard, and fibrous, with an excess of iron-pigment around the Malpighian follicle. All the lymph-glands were fibrous, and contained an excess of pigment. The kidneys were very fatty, whole areas of tubules were entirely degenerated—a condition difficult to harmonize with the absence of albuminuria. There was, likewise, excess of iron-pigment in the perivascular connective-tissue of the kidney. The pancreas and suprarenal glands presented no changes other than degeneration. The bone-marrow was a dark chocolate-red, a typical lymphoid marrow. Sections showed complete disappearance of the normal fat; the tissue consisted of large irregular marrow-cells filled with neutrophilic granulations, whole masses of normal leukocytes of all kinds, and many nucleated red corpuscles, of which a great many presented the different stages of cell-division.

I have searched carefully for sideriferous and globuliferous cells, but have not been able to find any. The brain and cord are in the hands of Dr. Sailer, who will later report to the Society the result of their study. I have here slides of the blood stained with the eosin, hematoxylin-mixture of Gallasch, the Ehrlich triple mixture, and dahlia; slides of marrow similarly stained; and sections of heart, liver, lymphatic glands, muscle, spleen, stomach, duodenum, pancreas, suprarenal capsule, kidneys, and the heart's blood, several slides of each stained with hematoxylin, thionin, and the carmin-ferrocyanid-hydrochloric stain for iron-pigment.

Discussion.—Dr. Judson Daland said he was extremely interested in Dr. Taylor's remarks on this condition. In reference to what would produce the loud murmur he described, the left ventricle was very much dilated, and it seemed to him that there must have been considerable regurgitation during life. The murmur was rather harsh and loud.

He remembered a case that occurred in the University Hospital, where the murmur was also very loud, and there was much doubt as to its origin. The patient had anemia, and the autopsy showed a heart similar to this. In addition to the dilatation and the intense anemia, as shown by the blood-examination, a muscular band was found stretched across the ventricular cavity. He had shown the heart here four years ago. An examination of the bone-marrow had not yet been thoroughly made. He was very much interested in the differential count of the leukocytes in this case.

About two years ago a case of pernicious anemia came under his observation, a patient of Dr. Seltzer's. The hemoglobin was three times the percentage of the red blood-cells. In this case repeated enteroclysis was carried out for three or four months, from one quart to three pints of liquid being used on alternate days. The only result observed was a slight improvement in the symptoms for a short period, and the possible prolongation of life for three months. Other than this there were no special results observed.

Dr. Alfred Stengel had seen a great deal of the patient. He first became a patient of his in September, 1894. He was quite anemic, having a little over a million reds, and 35 per cent. hemoglobin. He had typical pernicious anemia and was very ill.

He treated him with arsenic, with marked effect, and then gave arsenic and a glycerin-extract of bone-marrow, the extract being made from calves' ribs with glycerin. He got well enough to go back to his work. He was the driver of an ice-wagon, and often lifted 200 or 300 pounds of ice. He saw him sometime afterward, and he had red cheeks and every appearance of health, but upon examination of his blood he was surprised to find that he only had 45 per cent. of hemoglobin. His hemoglobin never came up to the normal, never being higher than 60 per cent. When he had the second attack he was brought to the city to the Howard Hospital. His corpuscles were down to 600,000, and his hemoglobin was about 15 per cent. He was slightly delirious and had to be strapped in bed. It seemed apparent to all that he must die, as he was running down steadily under arsenic. He was getting twenty drops of Fowler's solution three times a day, and then also bone-marrow, which was made by taking the bone-marrow from the tibia and mixing it with glycerin, making a sort of jam of it. It was very acceptable on bread, and he never tired of it. When this was begun and the arsenic kept up the improvement was startling, immediate, and progressive. In two or three weeks he was comfortable, and went home in a month. He came back in the fall, and was ill again, and was placed on the same treat-

ment. In spite of this and the arsenic and other regulations which he had had before, he ran down and died of asthenia. Dr. Stengel had no idea that bone-marrow was specific in this disease, but he began to think it was going to act so in this case. He wished Dr. Taylor had mentioned the ultimate nature of pernicious anemia. This man had seemed to him always to present the appearance of being a case of primary gastric or gastro-intestinal disturbance. The postmortem finding of fibroid changes in the stomach was certainly conclusive that this was not secondary to the anemia. It had been held that, although anemic changes are found in the stomach, they were secondary. Fatty degeneration such as existed in this case might be secondary to the anemia, but fibroid changes such as that man had, with the secondary changes in the tubules, could not be secondary to the anemia. It seemed to him that this was a case of primary gastric trouble. The stomach was not studied, and, in fact, he never had an opportunity to study him carefully.

In the case that he showed here a year ago the stomach was much more advanced in disorganization and in fibroid degeneration. In that case there was very much the same clinical history. The patient had been under treatment for stomach trouble and had been more or less ailing, with all the symptoms of gastric trouble. When she reached advanced age she began to fail progressively and became anemic, and this appearance of anemia never left her until her death. In that case there was advanced change in the stomach and all the characteristics of anemia.

In a recent case which he had had an opportunity of studying very little clinically but more elaborately postmortem, although as yet very little had been actually done, there were the blood-changes of pernicious anemia, and the lesion was cancer of the stomach with secondary involvement of the liver.

In another case which he presented to this Society two years ago, a case of Dr. Wilson's, there were the clinical symptoms of pernicious anemia, and the lesion, postmortem, was advanced gastritis with polypoid formations, not so numerous but quite as distinct as those seen in the interesting polypoid stomach presented by Dr. Hughes some time ago. This fourth case also presented advanced disorganization of the blood.

All these cases presented lesions of the stomach which could not well be attributed to the anemia itself. Without going into the question of how this might be traced to gastric or intestinal disturbance, he would simply like to make the statement that it seemed to him this origin of pernicious anemia had been well-established.

We must regard that condition of the blood as frequently due to

primary gastric disturbance, and that, if not all cases, certainly many originated in the gastro-intestinal region, more especially in the stomach.

Dr. A. E. Taylor said he had examined seven specimens of the blood, and only in two of them was there any marked degree of irregularity of the size of the cells. The important point in this case was that the hemoglobin was always considerably above the percentage of the corpuscles, hemoglobin 30 per cent., red cells 900,000. Before death, hemoglobin 22 per cent., red cells 13 per cent. With regard to the kidneys, they were white. Microscopically, one had to hunt for a healthy cell. The man never had albuminuria by any of the tests. How we might explain its absence was entirely unknown to him. *March* 12, 1896.

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12. *Chronic valvulitis and spinal caries.*

Exhibited by M. H. Fussell, M.D.

J. R., aged 45, male, native, died September 17, 1895.

Clinical diagnosis: Pott's disease and chronic endocarditis. *Pathologic diagnosis:* Pott's disease and organic disease of the heart, chronic parenchymatous nephritis.

Notes of autopsy. Body of a somewhat emaciated man, weight 101 pounds, rigor mortis well-marked.

Chest. Lungs can be seen in median line; left lung adherent at apex and by new adhesions; right, also; posterior mediastinal glands are not enlarged; pericardium contains three ounces of serum, straw-colored.

Heart normal size; left ventricle measures three-quarters of an inch in thickness; right ventricle one-quarter of an inch; cavities do not contain clots; the aortic valves are very much thickened.

The anterior leaflet contains two large heavy, calcareous deposits; the lateral, three, and the posterior, one large and one small.

The mitral valve is thickened, and the large calcareous deposit in the posterior leaflet of the aortic valve causes a prominence in the inside of anterior mitral. The mitral orifice merely admits the tips of two fingers.

There is a deposit in left lung in extreme upper apex, a scar of an old healed lesion, and some cheesy deposits. The entire upper portion of the left upper lobe is the seat of active infiltration.

Abdominal cavity. No presence of fluid; no adhesions; no miliary tubercles. Urinary bladder is distended with urine.

Mesenteric glands are slightly enlarged.

Spleen is enlarged, weighs 275 grams; no miliary tubercles.

Stomach is somewhat dilated; no abnormal deposit or growths.

Intestines abnormal, in that the walls are very thin.

Liver weighs 1900 grams; no tubercular deposits.

Gall-bladder is empty.

At the seat of spinal lesion, which begins at last cervical and extends to ninth dorsal, there is almost complete destruction of bodies of the vertebræ. At one spot opposite the fourth dorsal there is much destruction of the body, and the spinal cord with its envelopes is exposed.

Kidneys are large, and cortical substance largely in excess of medullary. *March* 12, 1896.

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13. *Aneurysm of the celiac axis, with atrophied heart.*

Exhibited by S. Solis-Cohen, M.D.

The patient was a colored man, about thirty years of age, a chimney-sweeper by occupation. He had been brought up to this work from his early boyhood. He acquired syphilis ten years ago. It could not be learned whether or not he had the ordinary diseases of childhood. The symptoms of which he complained had troubled him for about a year, but became acutely painful about two weeks before admission. The pains were of the nature that patients ordinarily ascribe to gastric derangements, nausea, discomfort after taking food, and slight pain in back. Careful examination was necessary to detect the pulsation of the abdomen, occupying an area about three inches in diameter in the left epigastrium and the left hypochondrium, that is to say, just to the left of the median line, the lower edge being about midway between the umbilicus and the border of the ribs. On careful palpation a thrill could be felt at a point which might be represented as the center of the palpating mass. The thrill was strangely limited, and to appreciate it the fingers had to be placed in a peculiar position, inclined diagonally from the left side upward. Auscultation over this region revealed a loud, systolic murmur, which could not be heard in the femorals. Percussion gave dulness at the same area, merging into liver-dulness. All attempts to outline the tumor on palpation utterly failed. Sinking deeply into the abdominal tissues an indistinct edge could be made out, and placing the forked fingers over the mass, one at each lateral limit, they could be observed to be moved further apart by the pulsations, showing the tumor to be

expansile. A diagnosis of aneurysm, probably at the celiac axis, was made in a clinical lecture at which the patient was exhibited. The patient was kept in bed, but from some unknown cause rupture of the aneurysm occurred, with prompt death, and at the autopsy the peritoneal cavity was found to be filled with blood. The rent is at the upper portion of the sac, which is exhibited. I was not present at the autopsy, but I believe the course of the blood was traced through Winslow's foramen into the general peritoneal cavity. The sac is connected with the abdominal aorta, but does not appear from a superficial examination to have originated in a distention of the aortic coat, though the orifice leading from the aorta into the sac is much dilated. Branches of the aorta above the celiac axis were found by dissection, but the celiac axis and its branches could not be found. I do not know whether the hepatic and gastric arteries were found at the other end, but I presume they were. The sac is not very thick, but large, not so large as my fist. The heart from the same patient is very much atrophied, is the subject of epicarditis with tortuous vessels, and presents the shiny, fibrous appearance common in hearts of this character.

I presume that the causes of the aneurysm were the patient's occupation and syphilis. The climbing and exposure must have caused a strain upon the vessels, while syphilis would tend to produce disease of the walls. The interest in the case was mainly diagnostic. Some say that unless a distinct mass can be made out a diagnosis of abdominal aneurysm is not justified, although one may find pulsation, thrill, and bruit. The specimen shows a bifurcating sacra media.

Dr. Sangree said he had witnessed a case very similar to this at an autopsy by the Coroner's physician, whom he had assisted. The man was a sailor who had suddenly fallen with severe cramps. At the autopsy there was blood in the abdomen on the left side, and an aneurysm was found almost precisely in the same location as Dr. Cohen's, and of about the same size. It also had an opening at the upper portion. The man had been syphilitic, as was shown by the spleen. The arch of the aorta was markedly atheromatous, although not calcareous.

Dr. Cohen said he should have stated, while speaking of the autopsy, that the liver partially overlay the mass, and this would account for the failure to outline the upper portion of the sac during life. The sac was filled with fibrin, and he could not be sure, from a rough examination of the specimen, whether or not there was an adventitious sac also. He imagined that the largest portion of the sac was adventitious, and that the real rupture occurred some time ago, the fatal hemorrhage having been a secondary one. *March* 26, 1896.

14. *The condition of the blood in the cachexia of carcinoma.*

Presented by Joseph Sailer, M.D., and A. E. Taylor, M.D.

In patients afflicted with cancer the more or less definite signs of the disease are generally dependent upon the locality of the neoplasm and the extent of its growth. However, cases in which no localizing signs are to be elicited are not rare, and it is in such cases as these that the study of the blood becomes of great importance. We have been enabled to study the blood of 22 cases of advanced cancer of various viscera; the diagnosis of these cases was confirmed either by necropsy or by histologic examination *in vivo*, or was so obvious that all doubt was excluded. Only the hemoglobin and the morphology of the blood could be studied. As venesection or venepuncture could rarely be performed, it was impossible to estimate the alkalinity and specific gravity of all the cases or to make chemical studies of the fibrin, sugar, salts, etc. For the material we are indebted to the kindness of Drs. Pepper, Stengel, Musser, White, Burr, Penrose, and Hand.

Case I.—Carcinoma of the stomach, with secondary nodules in the liver, lungs, kidney, and heart-wall, in a woman aged forty-five years.

Hemoglobin, 22 per cent.; red cells, 1,900,000; leukocytes, 22,000. There was considerable poikilocytosis, a few nucleated erythrocytes, all normoblasts.

Differential count of leukocytes. Neutrophilic polynuclear, 76 per cent.; oxyphilic polynuclear, 1 per cent.; mononuclear and transitional, 20 per cent.; lymphocytes, 3 per cent. No myelocytes could be found.

Postmortem the bone-marrow was that of a normal adult, the spleen and lymph-glands were normal, excepting for fibrous overgrowth, and there was no excess of iron in any of the organs.

Case II.—Carcinoma of very rapid growth in the stomach of a man aged fifty years, with a large tumor in the gastric area; symptoms positive. Cancer of pylorus, liver, and lymphatic glands was revealed by laparotomy done to form a gastro-intestinal anastomosis.

Hemoglobin, 30 per cent.; red cells, 2,812,000; leukocytes, 10,400. Cells were regular but pale, and none were nucleated.

Differential count. Neutrophilic polynuclear, 67.2 per cent.; oxyphilic polynuclear, 4.6 per cent.; mononuclear and transitional, 23.8 per cent.; myelocytes, 1 per cent.; oxyphilic mononuclear, 0.4 per cent.; lymphocytes, 3 per cent.

Case III.—Man, aged forty-five years, who died with signs and symp-

toms of pernicious anemia. A very small cancer, in no way proportionate to the symptoms, was discovered postmortem in the posterior wall of the stomach, with secondary growths in the head of the pancreas and in the retroperitoneal lymph-glands.

Hemoglobin, 20 per cent.: red cells, 1,100,000; leukocytes, 45,000; poikilocytes were present in moderation. There were some nucleated red cells, a few in nuclear division, which were all normoblasts.

Differential count. Neutrophilic polynuclear, 43.7 per cent.; oxyphilic polynuclear, 1 per cent.; mononuclear and transitional, 21 per cent.; myelocytes, 9.3 per cent.; lymphocytes, 25 per cent.

The marrow was less fatty than normal, and was lymphoid, only, however, in a few areas to a very slight degree. The spleen and lymph-glands were fibrous, the liver and kidneys somewhat fatty, and there was no excess of iron in any of the organs. While the symptoms were out of all proportion to the neoplasm, the morbid lesions of pernicious anemia were absent.

Case IV.—Carcinoma of the stomach, with interstitial nephritis, in a man, aged forty-seven years. Laparotomy for gastro-intestinal anastomosis showed cancer of pylorus, liver, and retroperitoneal glands.

Hemoglobin, 68 per cent.; red cells, 4,200,000; leukocytes, 3000; poikilocytosis was slight, and there were no nucleated red cells.

Differential count. Neutrophilic polynuclear, 88.7 per cent.; oxyphilic polynuclear, 2.5 per cent.; mononuclear and transitional, 5.0 per cent.; lymphocytes, 3.8 per cent. This count is entirely normal, except for the low proportion of lymphocytes, and in this man the symptoms were mild.

Case V.—Carcinoma of the stomach in a man aged sixty years. Laparotomy for gastro-intestinal anastomosis showed cancer of the posterior wall of the stomach and in the liver.

Hemoglobin, 55 per cent.; red-cells, 4,000,000; leukocytes, 10,000; poikilocytosis was moderate, and there were no nucleated red cells.

Differential count. Neutrophilic polynuclear, 66.5 per cent.; oxyphilic polynuclear, 1.6 per cent.; mononuclear and transitional, 14 per cent.; oxyphilic mononuclear, 0.2 per cent.; basophilic mononuclear, 0.2 per cent.; neutrophilic transitional, 0.2 per cent.; lymphocytes, 17.2 per cent. The neutrophilic transitional leukocytes were like the ordinary polynuclear cells in all respects except that the nucleus was large, and faintly and diffusely stained, and thus quite resembled some of the polymorphous leukocytes in the newborn.

Case VI.—Carcinoma of the rectum in a man aged fifty years, sections of which showed squamous carcinoma.

Hemoglobin, 25 per cent.; red cells, 2,400,000, leukocytes, 4000. There was a marked poikilocytosis of the red cells, which were very pale. There were many nucleated red cells of all kinds, microblasts, normoblasts, and gigantoblasts, and some free nuclei. Typical karyokinetic figures were seen in four large mononuclear leukocytes, and some of the nuclei of this type of leukocytes were fragmented.

Differential count. Neutrophilic polynuclear, 48 per cent.; oxyphilic polynuclear, 0.8 per cent.; mononuclear and transitional, 38.6 per cent.; myelocytes, 0.2 per cent.; lymphocytes, 12.6 per cent.

Case VII.—Carcinoma of the stomach, with secondary growths in the glands and omentum.

Hemoglobin, 46 per cent.; red cells, 3,600,000; leukocytes, 12,000. There was some poikilocytosis, but no nucleated red cells.

Differential count. Neutrophilic polynuclear, 76.5 per cent.; oxyphilic polynuclear, 1.0 per cent.; mononuclear and transitional, 12.5 per cent.; lymphocytes, 10 per cent.

Case VIII.—Carcinoma of the rectum in a man sixty years of age.

Hemoglobin, 38 per cent.; red cells, 4,000,000; leukocytes, 6000. There was considerable poikilocytosis, and some nucleated red cells.

Differential count. Neutrophilic polynuclear, 70.0 per cent.; oxyphilic polynuclear, 1.0 per cent.; basophilic polynuclear, 0.2 per cent.; mononuclear and transitional, 15.7 per cent.; lymphocytes, 13.0 per cent.

Case IX.—Carcinoma uteri; confirmed by sections after a later operation.

Hemoglobin, 30 per cent.; red cells, 3,600,000; leukocytes, 10,000. Some poikilocytosis was present; the cells were very pale, and there were a couple of nucleated red cells and quite a number of loose, fragmented nuclei of leucocytes.

Differential count. Neutrophilic polynuclear, 76.2 per cent.; oxyphilic polynuclear, 0.6 per cent.; mononuclear and transitional, 9.8 per cent.; lymphocytes, 13.4 per cent. Some of the mononuclear cells are very large and have huge nuclei ($10 \times 12\,\mu$), but the protoplasm is entirely homogeneous.

Case X.—Carcinoma situated near the cardiac end of the posterior wall of the stomach in a man of forty-eight years. Death consequent upon hemorrhage.

Hemoglobin, 25 per cent.; red cells, 2,000,000; leukocytes, 10,400. There were no nucleated red cells; but little poikilocytosis; many shadow-cells; no fragmented nuclei, and no karyokinetic figures.

Differential count. Neutrophilic polynuclear cells, 66.4 per cent.;

oxyphilic polynuclear, 0.4 per cent.; mononuclear and transitional, 8.8 per cent.; lymphocytes, 24.4 per cent.

CASE XI.—Carcinoma uteri in a woman of fifty-four years of age.

Hemoglobin, 23 per cent.; red cells, 1,856,125; leukocytes, 18,750. There were no nucleated red cells.

Differential count. Neutrophilic polynuclear, 76.5 per cent.; oxyphilic polynuclear, 1 per cent.; mononuclear and transitional, 12.5 per cent.; lymphocytes, 10 per cent.

CASE XII.—Carcinoma of the uterus and vagina in a woman of seventy years. Extreme cachexia.

Hemoglobin, 25 per cent.; red cells, 13,000; leukocytes, 25,000. There were no nucleated red cells.

Differential count. Neutrophilic polynuclear, 58.2 per cent.; oxyphilic polynuclear, 1.4 per cent.; mononuclear and transitional, 16.4 per cent.; lymphocytes, 24.0 per cent.

CASE XIII.—Carcinoma of the omentum in a woman of fifty-four years. Extreme cachexia.

Hemoglobin, 30 per cent.; red cells, 3,387,500; leukocytes, 18,600. No nucleated red cells were present.

Differential count. Neutrophilic polynuclear, 79.8 per cent.; oxyphilic polynuclear, 10.4 per cent.; mononuclear and transitional, 14.7 per cent.; lymphocytes, 5.3 per cent.

CASE XIV.—Cancer of the breast in a woman sixty years of age.

Hemoglobin estimation and corpuscular count were lost. There was moderate poikilocytosis, but no nucleated red cells.

Differential count. Neutrophilic polynuclear, 54 per cent.; oxyphilic polynuclear, 2.8 per cent.; mononuclear and transitional, 40 per cent.; lymphocytes, 3.4 per cent.

CASE XV.—Carcinoma of the scalp in a woman of sixty-three years.

Red cells, 3,900,000; leukocytes, 10,500. Poikilocytosis was present in moderation, but there were no nucleated red cells.

Differential count. Neutrophilic polynuclear, 66.3 per cent.; oxyphilic polynuclear, 0.2 per cent.; mononuclear and transitional, 28.8 per cent.; lymphocytes, 4.8 per cent.

CASE XVI.—Carcinoma of the left arm in a woman, amputation of which was followed by recurrences of cancer. Extreme cachexia.

Red cells, 4,385,000; leukocytes, 8000. Poikilocytosis was marked. There were no nucleated red cells.

Differential count. Neutrophilic polynuclear, 61 per cent.; oxyphilic polynuclear, 1 per cent.; mononuclear and transitional, 35 per cent.; lymphocytes, 3 per cent. This case illustrates the fact that the

appearance of extreme cachexia does not necessarily imply extreme anemia.

Case XVII.—Carcinoma of the breast in a female of fifty three years.

Red cells, 2,700,000; leukocytes, 9000. There was moderate poikilocytosis, and no nucleated red cells.

Differential count. Neutrophilic polynuclear, 61.2 per cent.; mononuclear and transitional, 34.8 per cent.; lymphocytes, 4 per cent.

Case XVIII.—Cancer of face, involving the bones, in a man fifty-eight years of age.

Red cells, 3,025,000; leukocytes, 6000; poikilocytosis was very marked, and there were many normoblasts.

Differential count. Neutrophilic polynuclear, 71.4 per cent.; oxyphilic polynuclear, 0.4 per cent.; mononuclear and transitional, 27.2 per cent.; typical myelocytes, 1 per cent.; lymphocytes, 10 per cent.

Case XIX.—Cancer of the colon in a male of sixty-seven years, who had undergone colotomy. He has marked cachexia.

Hemoglobin, 28 per cent.; red cells, 1,471,800; leukocytes, 14,800. There were moderate poikilocytosis and many normoblasts.

Differential count. Neutrophilic polynuclear, 66.2 per cent.; oxyphilic polynuclear, 2.5 per cent.; mononuclear and transitional, 27.9 per cent.; lymphocytes, 4.4 per cent.

Case XX.—In a woman fifty-one years of age, cancer of the uterus, in which the pelvis was markedly involved; confirmed by autopsy.

Hemoglobin, 37 per cent.; red cells, 2,190,000; leukocytes, 14,000; poikilocytosis was marked, and there were a few normoblasts.

Differential count. Neutrophilic polynuclear, 70.5 per cent.; oxyphilic polynuclear, 2.5 per cent.; mononuclear and transitional, 12.5 per cent.; oxyphilic mononuclear, 0.5 per cent.; lymphocytes, 14 per cent. Four myelocytes were noted.

Case XXI.—Carcinoma mammæ, with marked chest involvement, in a woman of forty-five years.

Hemoglobin, 48 per cent.; red-cells, 3,470,000; leukocytes, 14,600. There was marked poikilocytosis, but no nucleated red cells.

Differential count. Neutrophilic polynuclear, 68 per cent.; oxyphilic polynuclear, 0.4 per cent.; mononuclear and transitional, 18.5 per cent.; lymphocytes, 13 per cent.

Case XXII.—In a man aged forty-seven years, neoplasm in the right iliac fossa and in the liver; autopsy demonstrated cancer of the stomach and liver.

Hemoglobin, 37 per cent.; red cells, 3,260,000; white, 27,840; poiki-

locytosis was present in moderation; the cells were quite pale, and two were nucleated.

Differential count. Neutrophilic polynuclear, 65.6 per cent.; oxyphilic polynuclear, 0.4 per cent.; mononuclear and transitional, 27.3 per cent.; lymphocytes, 6.7 per cent.

The average of hemoglobin was 35 per cent., the extremes being 20 per cent. and 68 per cent., which is, however, the one estimation over 60 per cent. Between 50 and 60 per cent. there was one, between 40 and 50 per cent. there were two, between 30 and 40 per cent. six, and between 20 and 30 per cent. seven estimations. These figures, which were made with all precautions upon the Fleischl instruments, are much lower than the 45 to 65 per cent. estimation generally given (Laker,[1] Bierfreund,[2] Reinbach,[3] Rieder,[4] Cabot;[5] Harberlein[6] found a very low color index in gastric cancers). The cases were all advanced, but in only two instances was the anemia exaggerated by hemorrhage (Cases X. and XX., with 25 and 37 per cent. respectively). On examination the cases in the literature seem to divide themselves into three classes: a pseudo-chlorosis, of which we have not a good example in our list; or the blood may be quite normal or not markedly below normal, as in Cases IV. and XVI.; or, what is the most constant, moderate oligocythemia with more marked oligochromemia. The error with the Fleischl instrument is fully 10 per cent., which, however, appertains to the figures quoted as well as to our own. We believe that our error has been minimized by our having used several pipettes in the estimations; *e. g.*, when a preliminary estimation revealed a proportion of about 30 per cent., three pipettes would be used, in order to bring the reading up as near as possible to 100, in which case the error is obviously much less.

The average number of red-cells was 3,085,000, again rather lower than figures hitherto reported. Four cases were at or above 4,000,000; between 3,500,000 and 4,000,000 there were three; between 3,000,000 and 3,500,000, four; between 2,500,000 and 3,000,000, two; between 2,000,000 and 2,500,000, three; between 1,500,000 and 2,000,000, two; and between 1,100,000 and 1,500,000, three. The cells of normal shape were nearly always of normal size; apart from microcytes and macrocytes, we have not been able to determine any variation in the size of the cells in general. Their color varied with the hemoglobin; undeniable evidences of degeneration were never found. Microcytes and macrocytes were seen in varying proportions in nearly all cases, usually in limited numbers. As a rule, the macrocytes were disproportionately pale. Poikilocytosis was very variable: in several cases it was as extreme as we have ever seen it in true pernicious anemia, while in other cases it was

very slight. Nucleated red cells were seen in ten cases, in two in large numbers (VI. and XVIII.); in VI. there were microblasts and typical gigantoblasts. The total study of the red cells has not fully convinced us of the entire truth of the assertion of Strauer[7] that greater poikilocytosis is seen in cancer than is ever seen in tuberculosis. Polychromatophilia (which is not difficult to produce by fixing and staining manipulation) was rarely observed. The occurrence of nucleated red cells certainly does not indicate bone-metastasis. According to Loos[8] there is in infantile hereditary syphilis a peculiar polychromatophilia not seen in cases of cancer or tuberculosis.

Leukocytosis was seen in the majority (15) of our cases; it was doubtful in two (8000 and 9000) and entirely absent in four (3000, 4000, 6000, and 6000). The average in the cases of leukocytosis was 18,000. Leukocytosis in this condition is an old fact, having been pointed out in postmortem blood by Andral[9] and in living blood by Lücke,[10] previous to which pathologists believed the excess of the colorless cells to have been the cancer-cells in circulation, an error to which Paget[11] clung as late as 1870. It was well studied by Virchow. It has been found in the majority of cases studied by nearly all authors (Hayem,[12] Alexander,[13] Pee,[14] Schneider,[15] Rieder,[4] etc.), with the exception of cases of cancer of the esophagus with starvation, and of cases of superficial cancer, as on the skin of the face, the extremities, or the penis. Nor does it appear that the variety of carcinoma or the rapidity of growth has a constant influence, although metastasis quite generally heightens leukocytosis. Hemorrhage, as a rule, increases it. Apart from all these considerations, there are quite a number of cases which have no leukocytosis or have it inconstantly. Of our series, IV. (3000) had cancer of stomach with nephritis and moderate cachexia; VI. (4000) had cancer of the rectum (without obstruction), with marked cachexia; VIII. (6000), cancer of the rectum with some obstruction and moderate cachexia; XVI. (8000), thoracic recurrence following amputation of the arm, with marked cachexia; XVII. (9000), cancer of the breast, with moderate cachexia, and XVIII. (6000), cancer of the face and bones and moderate cachexia. In the cases of leukocytosis the highest, 45,000, 27,800, 25,000, and 22,000, were respectively in cases of cancer of stomach without metastasis, of abdomen and liver, of uterus and vagina with marked local extension, and of stomach with marked metastasis. From our cases, therefore, few conclusions upon this point can be drawn. The differential counts are very interesting. They show in general an increase in the nongranulated mononuclear and transitional varieties at the expense mainly of the lymphocytes. The average of the cases with leukocytosis was: neutro-

philic polynuclear cells 66.1 per cent., oxyphilic 1.5 per cent., mononuclear and transitional 19.3 per cent., lymphocytes 11.3 per cent. The excess of the monoculear and transitional forms over the lymphocytes is in striking contrast to normal blood. In only five of these cases were the lymphocytes in excess of the large mononuclear forms, and these cases are entirely overshadowed by the proportions in the remainder, of which seven showed less than 7 per cent. of lymphocytes. The same results hold more strikingly for the cases without leukocytosis, where the averages were: neutrophilic polynuclear 65.3 per cent., oxyphilic polynuclear 0.9 per cent., mononuclear and transitional 26 per cent., and lymphocytes 7.6 per cent. In not one of these cases were the lymphocytes equal in number to the large mononuclear cells. We have differentiated the lymphocytes from the mononuclear and transitional cells by the distinct difference in the appearance and staining of the nuclear chromatin, and by the relative proportions of nucleus and protoplasm. Our results are contradictory to the figures given by Reinbach[3] and Cabot.[5] The neutrophilic granulations in the polymorphous cells were less numerous than usually seen in the same variety of cells in septic leucocytosis. The oxyphilic cells were abnormally low, but that is a very common condition. Myelocytes were found in four cases; their presence in cachectic and infectious conditions has often been shown. We are not able to confirm the suggestion of Epstein[16] that the coexistence of myelocytes and nucleated red cells means bone-metastasis. Basophilic granulations were seen in two cases, and were of the fine variety. Amphoteric granulations were never seen. Rare changes in a few nuclei were observed in several cases, corresponding to the degenerations so well described by Gumprecht.[17] True karyokinetic figures were seen in one case, in large mononuclear cells. *Divisio per granulis* we were not able to demonstrate, but fragmented nuclei were often noted, as were free nuclei.

Müller[18] and Schneyer[19] believe that digestion-leukocytosis is absent in patients with carcinoma ventriculi. Hartung[20] found the same results; he also states that the injection of a nuclein-like substance, obtained from the digestion of calves' spleens, produces no reaction of leukocytosis in cancerous subjects, whereas the reaction follows such injections into non-cancerous subjects. We have not been able to study this question. Hausmann[21] and Capps[22] have observed digestion-leukocytosis in cases of gastric cancer, while, on the contrary, we have noted an entire absence of digestion-leukocytosis in a case of tubercular cachexia and in two cases of infectious leukocytosis. Before a definite conclusion can be reached it must first be determined whether and to

what extent a proteid meal produces a further increase in the leukocytes in patients suffering with leukocytosis, and also as to how extensive is the influence of pyloric obstruction and gastrectasis.

We are inclined to the belief that an excess of large mononuclear leukocytes as against lymphocytes is of distinct value in the diagnosis of carcinomatous cachexia in distinction to pernicious anemia; as against tuberculosis it is less suggestive.

The chemical studies of the blood in cancer have not been elaborate or complete. The specific gravity has been found reduced by Deveto,[23] Peiper,[24] Schmaltz,[25] Schalkoff,[26] Moraczewski,[27] Grawitz,[28] Strauer,[7] and others, not always by the reliable method of weighing. In two of our cases (II. and IV.) estimations were made by filling a 5-gram picnometer with blood from several deep lance-punctures in the ring-finger, and weighing; the figures were 1047, 1048.7, respectively. The diminution seems to be caused only partly by the oligocythemia and partly by a hydremia of the plasma (von Jaksch).[29] The same author has noted a reduction in the nitrogen, but this cannot without more data be attributed entirely to a hypalbuminosis, which Bleibtreu[30] could not confirm. Biernacki[31] believes that hypalbuminosis exists, especially in the red cells. He studied the salts and found a moderate diminution in the iron and a more marked decrease in the potassium and in the phosphates. (In his later work he denies that the iron is at all diminished.) There seems to be an increase in the chlorids, both in the plasma and in the tissues (Landenberger,[32] Schäpp,[33] Moraczewski[34]). Just what relation this fact bears to the relative proportion of sodium and potassium, the gastric anacidity and defective alimentary absorption, and the tissue hydremia and increased nitrogenous disintegration, as revealed by a disproportionate output, is very obscure.[35] The facts are not peculiar to cancer. The dried residue is much reduced.[36] Müller[37] and Grawitz[28] believe that there is a decided hydremia (denied by Hammerschlag[38]), which Grawitz believes is due not solely to metabolic and nutritional disturbances, but also to the lymphogoghic action of some toxic substance, and he has detailed experiments in which cancer-extract was injected into rabbits, resulting in the production of hydremia and the diminution of the specific gravity and dried residue—just the opposite to what followed the injection of tuberculous extracts. The alkalinity has often been described as decreased, but, with the exception of the cases of Klemperer,[39] who found the CO_2 greatly diminished, the methods have not been accurate. There is, however, evidence that both organic and inorganic acids are increased, with increased basic capacity.[35] Freund[40] in a large series of cases described an excess of sugar in the

blood, and also noted a reduction after operation (analogous to the reduction in leukocytosis after operation), and his results have been confirmed by Trinkler.[41] Matfaie,[42] however, denies that this is constant or characteristic.

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Discussion.—Dr. Burr thought the Society ought to congratulate itself on having heard this very interesting paper.

Dr. Stengel was pleased that Drs. Sailer and Taylor had done this work, because those who had done anything of this kind knew how very

laborious it was, and the results which they got were so positive in contradicting and successfully refuting the claim of Neusser that the myelocyte was an indication of cancer. He himself had made differential counts of the blood of comparatively few cases of cancer of the stomach or of other situations, and had no experience in that direction; but he had frequently found myelocytes in diseases other than pernicious anemia, in leukemia, or in localized osseous disease.

His idea of them—as it has formed rather slowly, and was not very satisfactory even now—was that the myelocytes were cells, either originating from the bone-marrow, or in some way influenced by it; that they were found in various diseases, according as the bone marrow was affected or not. In the autopsies in cases in which they had found myelocytes, it was impossible to assert that the bone marrow was or was not affected; of course the bone-marrow was most extensive, and could not be examined in its entirety, and even if it could be so examined, it could not be said that the red marrow had not overrun its normal limit and had involved parts which should not normally be involved.

Of course, there was one thing to be said, that in the pathologic transition of lymphoid marrow the transition began in the long bones of the periphery, and, therefore, we were able to control and determine how much red marrow there was.

As long as it was impossible to determine when the bone-marrow was affected, it was impossible to decide in what classes of diseases, or to what extent in various diseases, cells originating from the bone-marrow, normally or pathologically, would be found. The accounts published by Cabot had struck him particularly. While he believed that Cabot had looked upon cells as myelocytes which were probably not myelocytes (because he obtained such very remarkable and striking counts of myelocytes), yet his results certainly indicated that there were a very large number of diseases in which myelocytes could be found. Investigations had shown that in almost all infectious diseases, such as erysipelas, the bone marrow was involved to a certain extent. Now such cases, if we counted a similarly large number of cases, as Drs. Sailer and Taylor had done, would show, perhaps, an equally excessive number of myelocytes.

He believed that the value of the Neusser sign, as it might be called, was absolutely nil; he would not concede to it the importance that had been given to it by Drs. Sailer and Taylor. He was interested in the study of the blood as a diagnostic indication of cancer on two occasions particularly: at one time when a case of tumor of the mediastinum was under observation, and it was of interest to decide whether it was carcinomatous or sarcomatous; and in the second place, on several

occasions when patients presenting the features of pernicious anemia had been under observation and had presented very marked gastric symptoms.

When a hospital resident in Dr. Henry's wards he had had just such a case: an old man presenting all the features of pernicious anemia, in which the ordinary blood-count was made, and in which the general appearances of the patient were those of pernicious anemia, and the stomach symptoms were pronounced. There was a very marked oligocythemia, and a very moderate oligochromemia; there was almost twice as much hemoglobin as there should have been for the number of corpuscles. That, he believed, made the diagnosis between those two conditions absolute. The case afterward came to autopsy, and the diagnosis was confirmed in that way.

In one of the cases that Dr. Taylor reported, in which, as he put it, the condition of the patient, the blood, the cachexia, were out of all proportion to the extent of the carcinoma, it was difficult to say that this was a simple carcinomatous cachexia; his belief was that it was a case of pernicious anemia. Cases of pernicious anemia in which the only pathologic lesion found was carcinoma, usually of the stomach, sometimes of the intestines, had been reported from time to time. Only recently Lubarsch had reported four, in which the physical symptoms and the blood pointed to pernicious anemia, and cancer was the lesion.

In the particular case referred to, while it was not seen until just shortly before death, still the appearance of the patient, the relative excess of coloring matter, and the condition of the corpuscles, made him believe that it was really a case of pernicious anemia; that, while the man had carcinomatous cachexia, he was suffering from those very obscure changes that occurred in the gastro-intestinal tract, and led to that condition of the blood which we called pernicious anemia.

In cases where the diagnosis of carcinoma of the stomach and pernicious anemia was under discussion, the blood-count would be important. His own counts of the blood had shown invariably, as Dr. Taylor had stated, a high grade of reduction of coloring matter without very large reduction of the number of the corpuscles; indeed, in the cases of the most extreme apparent anemia, in cancer, the number of corpuscles sometimes had struck him as simply marvellous, 4,000,000 to 5,000,000, although usually the counts had run lower than that. The lowest count he had ever seen in carcinoma patients, without suspicion of pernicious anemia, was a million or thereabouts. Cases had been reported with a count of 500,000; Limbeck saw a case of cancer in which the count ran down to 500,000, but the question was whether that was a pure

case of cancer, and it seemed more probable that it was a case of hemorrhage.

As far as leukocytosis was concerned, either as a constant thing, or as postdigestive, or as a dietary leukocytosis, he felt that there was no value to be attached to it as an indication of carcinoma, and this was the general view. Some few, however, had claimed for this a peculiar value. If one had counted the blood of a number of cases after meals, it would strike him that a large proportion did not show enough postdigestive leukocytosis to be noteworthy, not enough to decide. After all, the normal number of leukocytes in the blood was far from settled, and the slight increase that might occur in the blood after a meal was not sufficient to make a diagnosis.

Obstruction of the pylorus acted as a mechanical thing. This had been shown by Boas' test for lactic acid in the stomach. Of course, there were differences. He did not feel that we were getting at useful results by counting the blood of carcinomatous patients after meals, with the hope of finding a less number of leukocytes.

Dr. Sailer said that he had entered upon this study with a very strong prejudice in favor of the view that there was a peculiar physiognomy, as it were, of the blood of carcinoma. He had formerly seen a number of cases in which this seemed to be pronounced, and the first case to which his attention was called in this city, the extraordinary case from the German Hospital, where the myelocytes equalled 9 per cent. of the total number of white corpuscles, seemed very strongly to confirm this view. But, in the course of the counts he had made his opinion had entirely changed. He believed now that there was no such physiognomy; that the blood of cancer, as a rule, showed nothing to differentiate it in any way from the severe secondary anemia; and that the myelocyte was exceedingly elusive and hard to discover. The most characteristic feature, aside from the unusually large number of simple mononuclear cells, was the diminution of the eosinophile cells. This was certainly remarkable, considering the number of corpuscles counted and the amount of leukocytes. He expressed his and Dr. Taylor's obligations to the residents of the Philadelphia Hospital for their courtesy in giving them every facility for the study of the blood of the number of cases of carcinoma in that hospital.

Dr. Eshner recalled a recent report of a case of widespread carcinosis involving also the bone-marrow, in which, during life, he believed no blood-counts were made, but at the autopsy such observations were taken, in which such relations of blood coloring-matter and blood-cells were found as constituted the picture of pernicious anemia. He did not

remember the exact figures, but thought the number of red cells was about a million to a million and a half, and the proportion of hemoglobin was rather above that. In this instance special stress was laid upon the predominance of the leukocytes; the proportion averaged from 1 to 25, to 1 to 40.

Dr. Taylor said that Ehrlich gave four reasons for his view. The first was, that the typical cell divided by karyokinesis; the large mononuclear cells were practically never seen in karyokinesis. Then they did not possess movement. The large cell had very little ameboid movement, but it had some. The myelocyte of Ehrlich possessed no movement at all. It contained neutrophilic granulations, which were never seen, according to Ehrlich, in ordinary mononuclear cells. The fourth reason concerned itself with the distribution of the fibers in the nucleus.

It seemed that the line along which work must be done in differentiation of these cells was a totally different line. It had been shown that there existed, especially in young cells, a system of central bodies, and of fibrillæ which ran from these central bodies through the nuclei into the protoplasm, connecting the whole cell into one system of fibers, and that the cell was not divided into a nucleus and protoplasm. Ernst had shown that the eosinophile granulations were part and parcel of those fibers. He had shown this especially for the large giant-cell of the bone-marrow. In the nucleated red corpuscles of the embryo there was one stage when the cell was filled with basophile granulations. With the reproduction of the nucleus, and at the extrusion of this set of fibers, the granulations disappeared. If that could be shown to be true of adult blood, it would be absolutely certain that the myelocyte of Ehrlich was not identical with the bone-marrow cell.

May 14, 1896.

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15. *Localized endocarditis of the tricuspid valve.*

Exhibited by Frederick A. Packard, M.D.

The specimen was removed from the body of a male German giving the history and physical signs of acute catarrhal pneumonia. On the anterior tricuspid leaflet, projecting above the general level to a height of 3 mm., there is a tumor-like sessile mass of circular shape, measuring 11 mm. in diameter; it is white and soft. In the recent condition it was capped by a layer of white fibrin. Its surface is roughened, and its base

is surrounded by a sort of bank apparently composed of thickened endocardium; the surface looks as though ulcerated. On the posterior leaflet of the tricuspid valve, at a point corresponding to the position of the other mass, there is a small papillary outgrowth, 5 mm. in length, 3 mm. in height, and about 5 mm. in width; its surface is rough, its color reddish. *June* 11, 1896.

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16. *Tricuspid and mitral stenosis; persistent ductus arteriosus; pulmonary thrombosis.*

Exhibited by Frederick A. Packard, M.D.

The patient from whom this heart was obtained was under my observation, from time to time, for a year before his death. He was thirty-five years of age, a one-legged pugilist by occupation. When I first saw him I was temporarily in charge of the men's medical ward at the Pennsylvania Hospital, to which institution he was admitted with pain at the precordium after drinking an excessive amount of beer. He then had the signs of acute dilatation of the heart, which appeared to subside after two or three days of treatment. Eight months later he again came under my care with partial pulmonary consolidation of the right lower lobe, great increase of the area of cardiac dulness, a loud mitral systolic murmur, well transmitted to the left axilla; another harsher systolic murmur of lower pitch heard over the body of the heart, and a soft systolic aortic murmur. The liver was somewhat enlarged. After a few days in the hospital he had to be discharged for insubordination. Before a week had elapsed he was brought to my ward at the Philadelphia Hospital, having been brought in from the street on the ambulance because of a sudden choking sensation with a profuse expectoration, first of bloody mucus and then of pure blood. He had evident signs of pulmonary infarction of the right lower lobe. The whole left chest anteriorly seemed to heave with each cardiac contraction. There was a presystolic thrill just within the apex. Cardiac dulness extended from the right parasternal line almost to the midaxillary line of the left side, and from the third interspace above to the sixth interspace in the nipple line, the seventh at the apex. At the apex there were found a short, soft presystolic murmur not transmitted, and a soft blowing, but rather high-pitched, systolic murmur transmitted to the left axilla and heard at the left scapular angle. In the fifth interspace, just to the left of the sternum, there was well-heard a high-pitched systolic murmur distinctly

differing in pitch and quality from that heard at the apex. After a few days rest in bed, with cardiac treatment, the heart quieted down, and the following note was made of the heart-sounds: At the apex over an area covered by two bells of the stethoscope a long rumbling presystolic murmur filling almost the whole diastole, and followed by a blowing systolic rather low-pitched and well-transmitted systolic murmur. Both sounds at the aortic area feeble and unaccompanied by murmur. Over the body of the heart in an area covered by three bells of the stethoscope there is a high-pitched, well-defined systolic murmur, differing in character and pitch from the apical systolic murmur. This condition continued for about three weeks, when he again became unruly and had to be discharged. About four months later, when I happened to be again temporarily in charge of the wards of the Pennsylvania Hospital, he applied for admission for extreme dyspnea and cyanosis. The heart was in almost the same condition as before. His symptoms of failing compensation steadily increased until his death. At autopsy the following condition of the heart was noted: The pericardium was partially united by long adhesions of varying degrees of thickness containing in their meshes much blood-stained serum. The weight of the heart freed of pericardium and clots was thirty-six ounces. The heart was globular in shape, the apex being made up of the left ventricle, but the septal line being very near the point. The right auricle was much dilated, the auricular appendix blunt and squared. The thickness of the right auricular wall in places equaled 5 mm. Foramen ovale closed. The endocardium of the right auricle thickened and opaque, in spots more markedly than over the general surface. On looking into the tricuspid opening from the auricle the upper orifice was seen to be larger than the lower. The tricuspid leaflets were welded together, forming a cone-shaped wall. The orifice between the edges of the welded valve-leaflets admitted two fingers with great difficulty. The auricular surface of the leaflets was roughened, but not ulcerated or calcified. From the ventricular side the leaflets were seen to be welded together, forming a uniform continuous boundary to the tricuspid funnel. The chordæ tendineæ of the tricuspid were somewhat thickened, while the valve-leaflets themselves were very opaque and much thickened. The right ventricular wall measured 3.8 mm. in thickness, the muscle being somewhat pale. The endocardium of the general surface of the right ventricle looked fairly normal, but at the base of the pulmonary leaflets in the conus arteriosus and near the apices of the papillary muscles there are white, opaque areas of endocardial thickening; ventricular septum complete. The pulmonary valve-leaflets are very large, slightly opaque, and the posterior leaflet fenestrated. The

orifice of the pulmonary artery measures 11.5 cm. The artery rapidly narrows so that at a point 2 cm. above the base of the valve-leaflets its circumference is but 9 cm. The wall of the artery is decidedly thicker than normal. Division of the artery takes place 2 cm. above the origin of the leaflets. The remains of the ductus arteriosus are plainly felt as a solid cord uniting the aorta and pulmonary artery. Through 13 mm. of its course a bristle could be passed from the former vessel to the lining membrane of the pulmonary artery. The left auricle was tremendously dilated, its wall thin (too thin for measurement). The auricular appendix was much dilated, blunt and square in shape. The endocardium of the auricle was opaque and evidently thickened. From the auricular aspect the mitral orifice showed itself as a straight slit, bounded by thickened, well-defined wall formed by fused leaflets, with calcification. In one place at the anterior end of the slit there was a rough area devoid of endothelium, with projecting plates of calcareous material. The lower orifice of the mitral funnel admitted one finger. The left ventricle was greatly dilated, its apex rounded, and its wall measured 11 to 13 mm. in thickness; the muscle was a little pale. The endocardium was quite opaque over the region just below the aortic leaflets and at the apices of the papillary muscles. The chordæ tendineæ were much thickened and opaque, stiff and welded together. The aortic leaflets were thickened and opaque, the adjacent cusps being welded together. The circumference of the aortic orifice was 7 cm. (much less than that of the pulmonary artery). The lining membrane of the aorta was smooth, and 8 cm. above its root was seen the orifice of the ductus arteriosus.

In a branch of the pulmonary artery, 3 mm. in diameter, running to the upper lobe of the right lung, there was seen a thrombus completely occluding the vessel and running into quite a large branch.

June 11, 1896.

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17. *Fibrinous pericarditis with hypertrophy and dilatation of the heart; partial patulousness of ductus Botalli.*

Exhibited by David Riesman, M.D.

This specimen of heart and lungs was removed from a young girl, fifteen years old, who had been under the care of Dr. Longaker. She was first seen in 1894, during convalescence from an attack of acute articular rheumatism, and was found to have an apical systolic murmur; she had also chorea at that time. For nearly two years she was not under treat-

ment; last March Dr. Longaker was called to attend her again, and found her suffering from right-sided lobar pneumonia. This was slow in resolving, but eventually did so, and was followed by general joint-pains and muscular soreness, associated with profuse sweats. She complained of precordial pain and tenderness to pressure; the cardiac area was enlarged, and symptoms of failing compensation set in. The girl was also excessively anemic. Death took place on Friday. The autopsy was made on Saturday.

The body was that of a young woman of normal height, but with under-development of the breasts and external genitalia. Percussion of the precordia before opening chest showed the upper heart-boundary in second interspace, lower at seventh rib, and right boundary at right border of sternum. The subcutaneous tissue and the muscles generally were edematous. There was a large amount of fluid in the peritoneal cavity; the liver was much enlarged and projected four inches below the costal border. The diaphragm on the right side extended to the top of the fifth rib, on the left to the top of the sixth. The mediastinal tissues were edematous. A considerable amount of thymus gland was found remaining. The cardiac boundaries coincided practically with the area mapped out, except that the organ extended a little further to the right. Recent adhesions bound the pericardium and pleura together. There was a large amount of slightly bloody fluid in the left pleural cavity; a lesser amount in the right. The lungs were considerably compressed, but still contained some air; the apices and edges were emphysematous; the anterior lower edge of left lobe was intensely edematous; there were recent diaphragmatic adhesions on the right side. The pericardium was the seat of most marked fibrous pericarditis gluing the two layers together throughout their extent. They were separated, however, without difficulty, and left a surface comparable to an ox-tongue; the projections were irregular, rough, shaggy; they were largest over the right auricle; in places the uniting bands were of considerable length. Small hemorrhages were scattered over the two surfaces, and there was a little slightly bloody fluid in what remained of the pericardial sac. The deposits extended up along the aorta. The heart was globular in shape. On dissecting up the great vessels a connecting band or cord was found between the aorta and left pulmonary artery—it was the *ductus Botalli* or *ductus arteriosus*. It was 12 mm. long, and arose from the lower edge of the arch and ran to the top of the left pulmonary artery, joining it at a point 2½ cm. before the entrance of the vessel into the hilus of the lung. Its origin corresponded to a point 1 cm. beyond the left subclavian artery, and was about 2½ cm. from the root of the aorta. It was 4 mm. in diameter, and felt like a cord, especi-

ally in its upper part. A probe could not be introduced from the aorta, and there was here no sign of opening, but from the pulmonic side the probe entered a short distance, proving that the duct had not entirely closed. The heart showed hypertrophy and dilatation of the cavities. On the surface of the right lung were small, translucent, whitish elevations, looking like miliary tubercles. They were, however, not tubercles, but probably represented dilated lymph-spaces. The glands of the superior mediastinum were enlarged, but not cheesy.

The *kidneys* were pig-backed and firm, the capsule slightly adherent, the surface dark. The cortex was somewhat reduced in thickness, and it, as well as the pyramids, was congested.

The *liver* was enlarged, the edges rounded, its consistency increased. On section it was typically nutmeg, and showed some fatty change.

The *cecum* was only an inch in length, while the appendix was unusually long. The mesenteric glands were all enlarged, but presented no evidence of tuberculous change.

The *uterus* was infantile; the left ovary had hanging from it a small, fibrous pediculated body.

On microscopic examination we find that the pericardium is covered with a deposit of fibrin, and is infiltrated with round cells, and in places shows minute abscesses, which clearly prove the infectious nature of the process. In a section stained with thionin I believe I found some micrococci. There are also many epithelioidal cells, showing that organization was in progress. The liver shows congestion of the central veins and the intralobular capillaries, with moderate pigmentation and fatty infiltration. The kidney presents an intense degree of cloudy swelling, and some increase of connective tissue about the glomeruli, but on the whole much less than the adhesions of the capsule noted at the autopsy had warranted us in expecting.

A few words will not be out of place in reference to the partly patulous ductus Botalli. This canal, as you know, in the fetus conducts most of the blood poured by the heart into the pulmonary artery into the aorta, and joins the latter, as in this specimen, at its concavity beyond the left subclavian artery. It is formed by the junction of the fifth aortic arch on the left side and the fourth arch. Normally it begins to close immediately after birth, and by the tenth day is usually closed, and its lumen is completely obliterated by the third week. Closure usually starts at the aortic end. The process of obliteration, as shown by Parrot and also by J. Collins Warren in a lecture before this Society, depends on a proliferation of the media, the inner coat being thereby thrown into folds. The endothelium also proliferates and projects into the delicate fibrinous mass which

forms in the narrowed channel of the vessel. Under certain conditions obliteration does not occur; the duct may then be patulous throughout, or it may be open at the pulmonary or at the aortic end, or, which according to Moussous is more frequent, it is open at both ends, while the center is converted into a fibrous cord. Its dilatation is sometimes so great that it is with difficulty distinguished from the pulmonary artery. In rare cases it has been double.

The causes of nonclosure are not definitely known. In some cases patency is associated with other vices of conformation on the part of the heart, but at times it is the only anomaly present. It has been ascribed to the existence of some obstruction, such as atelectasis of the lungs; premature delivery has also been assigned as a cause. In one case, reported by Peacock, the duct was entirely absent. In hastily scanning the literature I find quite a number of instances recorded in which the duct was patulous; one reported by Luys was a patient, aged fifty-two—the duct admitted the little finger; Fagge's case was forty-five years old; Peacock's, thirty; Hochhaus', twenty-four; Bernutz's, twenty-three. These ages show that the anomaly is not one that necessarily leads to early death. The diagnosis is, of course, difficult, but seems to have been made in Hochhaus' case, with verification at autopsy, and by François-Frank. There is usually a systolic, rarely a diastolic murmur, which has its maximum intensity at the pulmonic cartilage, is not transmitted for any distance in front, but is distinctly audible in the back, between the left scapula and the upper dorsal vertebral spines. The murmur is increased during inspiration and decreased during expiration. The first and second pulmonic sounds are clear. The pulse is fuller during expiration than during inspiration. Cyanosis is either entirely absent or very slight. Carpenter states that a persistent duct is very liable to atheroma. Aneurysmal dilatation is also said to occur, as perhaps in the case of Bernutz.

Another point of interest in our case was the profound anemia. Rheumatism is, of course, capable of producing a grave anemia. If we add to this the pneumonia it is not surprising that the pallor of the girl was extreme. But having in mind a case that I saw when a hospital resident—a patient of Dr. Packard's—in whom the blood-count was that of pernicious anemia, and the heart almost like this one, the question has recurred to me whether there is usually an extreme anemia in the cases of fibrinous pericarditis. If it exists it does not necessarily follow that the pericarditis aggravates the anemia. Perhaps the only justifiable inference is that both the anemia and the pericarditis are an expression of an intense degree of infection.

Discussion.—Dr. Longacker was most impressed by the long history of rheumatism, the profound anemia, and the rapid development of the dilatation of the heart.

Dr. F. A. Packard mentioned the fact that Dr. Griffith had recently made a very beautiful diagnosis in a case of congenital heart disease, consisting of patulous ductus arteriosus. *June* 11, 1896.

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18. *Atheromatous ulcer of the tricuspid valve.*

Exhibited by J. Dutton Steele, M.D.

The following case was admitted to the wards of the Philadelphia Hospital under the care of Dr. John H. Musser in the spring of 1896: The patient was a male, aged seventy years, white, a laborer by occupation. There is no tubercular, rheumatic, or alcoholic family-history. He had the ordinary diseases of childhood; was a moderate drinker; denies specific history; has never had rheumatism; had always been a fairly healthy man up to the time of admission. He came to the hospital for increasing shortness of breath and general muscular weakness. He had always been employed at hard manual labor.

Physical examination showed the heart to extend to the right midclavicular line and to the left beyond the midclavicular line. The pulse was slow, 60 to 70 per minute, and of high tension. There was a loud systolic murmur in the aortic area transmitted to the bloodvessels of the neck. The second sound was accentuated. At the apex the first sound lacked muscular force. No tricuspid murmur was detected. The expansion was poor over both apices; the breathing was harsh, and there were a few dry râles. The urine, on admission and up to the time of death, contained a small quantity of albumin and varied in amount from 25 to 30 ounces in twenty-four hours. His temperature was persistently subnormal, and four days before death sank to 93.4°. It rose quickly to 100°, then to 102°, and then gradually sank to 100.2° at death. He gradually failed after admission and died with well-marked symptoms of uremia.

Autopsy. The third toe of the right foot is gangrenous. There are several old cheesy nodules at the back of the left lung. There are very extensive and atheromatous changes in all the arteries examined. The heart is much enlarged and weighs 500 grams. The coronary arteries are much diseased and distended, being as large as a cedar lead-pencil

at their beginning; they are tortuous and show commencing calcareous infiltration. The muscle of the left ventricle measures 25 mm. in thickness; that of the right, 6 mm. The heart-muscle is pale and friable, with many areas of fibroid change. There is a calcareous nodule in the wall of the left ventricle, in the auriculoventricular ring behind the insertion of the posterior leaflet of the mitral valve. The aorta is exceedingly atheromatous, and the aortic valves inside and out are studded with calcareous nodules. There was very little evidence of acute inflammation or impairment of function. The tricuspid valve shows considerable fibrous change around its edges.

The posterior leaflet is particularly affected, and on its under surface there is an ulcer with ragged edges, but there is no evidence of acute inflammation about it.

Microscopic examination of this ulcer shows it to be the result of fatty degeneration. The valve is much thickened by fibrous tissue, which, around the ulcer, shows much granular material that increases in amount as it approaches the floor of the ulcer. There is much granular *dèbris* over this floor, but a remarkable absence of all inflammatory action. The ulcer was evidently formed by the breaking away of a mass of fatty material that had formed in the connective tissue of the atheromatous valve. The mitral is somewhat thickened, but not as extensively as the tricuspid. The pulmonary valves are normal. There are cirrhosis and cyanotic induration of the spleen, liver, and kidney.

June 11, 1896.

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19. *Great cardiac hypertrophy, without valvular lesions.*

Exhibited by J. Dutton Steele, M.D.

The following specimen is shown because of the very extensive hypertrophy of the myocardium unaccompanied by valvular lesions. The explanation of the great increase of the muscle is doubtless to be found in the chronic interstitial nephritis and extensive arteriocapillary sclerosis that is present.

C. L., male, white, a German, aged forty-nine years. A large, well-developed man. There was extreme edema of the abdominal wall and lower extremities. The pleural sacs were both full of fluid. The lungs were edematous and congested.

Pericardium. There are remains of an old pericarditis over the surfaces of the left and right ventricle, with no adhesions.

Heart. Enormously enlarged and somewhat spherical in shape. The right auricle is greatly dilated. The left ventricle contains marantic clots. It measures 25 mm. in thickness, 11½ cm. in length. The right ventricle measures 10 mm. in thickness and 15 cm. in length. The aortic valves are competent and their endocardium is normal. The mitral valve admits three fingers, and there are a fibrous thickening and several spots of atheroma at the base of the valve, but they are not incompetent and not sufficiently diseased to offer obstruction to the blood-current. The myocardium is somewhat pale, but there are no areas of interstitial change.

The papillary muscles especially are pale, and have the yellow tinge of commencing degeneration. The tricuspid valves are dilated, and admit five fingers and thumb.

The right auricle measures 7 mm. in thickness; the left auricle, the same. The valves of the right side are perfectly normal. The weight of the heart is 790 grams.

The spleen, liver, and kidneys show some cyanotic induration.

June 11, 1896.

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20. *Chronic endocarditis associated with pulmonary tuberculosis.*

Exhibited by Joseph Sailer, M.D.

In the course of about sixty autopsies at the Philadelphia Hospital since April 1, 1896, three presented the complication of chronic endocarditis and pulmonary tuberculosis, and as the proportion seemed unusually large, it seemed worth while to record them.

M. A. G., white, female, aged sixty-seven years; greatly emaciated. The pathologic diagnosis was carcinoma of the stomach, cardiac end, mitral obstruction, tuberculosis of right apex. In the right apex there was a dense fibrous mass, quite solid, and another showing necrosis of its center.

H. F., white, male, aged fifty-one years. Well-nourished; lower extremities edematous; ascites; mesenteric glands enlarged; pleural adhesions.

Left lung: A patch of consolidation containing tubercles on either side of the interlobular septum.

Right lung: Middle lobe consolidated, containing tubercles and three cavities, about the size of a walnut, filled with pus.

Spleen firm and enlarged. Chronic interstitial nephritis with paren-

chymatous change. Tuberculous ulceration of the ileum and ileocecal valve.

Heart: Calcification of postmitral leaflet, with obstruction of mitral orifice; atheromatous changes of the aortic valves.

S. D., aged sixty-one years, white, female. Moderately well-nourished. Pleural and pericardial adhesions. Chronic interstitial myocarditis; edges of mitral valve thickened, with numerous patches of atheroma in the valves. Sclerosis of the aortic valves.

Right upper lobe completely infiltrated, containing a large cavity with necrotic walls. Disseminated tuberculous infiltration of middle and lower lobes, large tuberculous nodule in the lower lobe.

Left lung: Slight infiltration of the upper lobe near the interlobular septum with tubercles. Chronic interstitial nephritis; large irregular ulcers in the cecum.

Weismayr has recently reported 6 cases of valvular lesions of the heart associated with tuberculosis. He has further collected some interesting statistics. Frommolt found 21 cases of tuberculosis in 276 of heart-disease. Kidd found the combination 27 times, in 11 the heart lesion antedated the tuberculous condition. Kryger found 10 cases in 1100 autopsies, and 59 cases of valvular lesion and healed tuberculosis. Potain found 9 cases of tuberculosis in 59 of mitral stenosis. In Weismayr's own cases, 4 had the valvular lesion before the appearance of tuberculous symptoms.

Of the 3 cases reported, in 1 the tuberculosis had evidently undergone fibrous change, and was latent; in 1 the heart was so slightly affected that very little congestion could have resulted, and death was caused by an independent affection. In the third, a very chronic cardiac lesion of many years' standing was associated with a recent tuberculous process that caused death. *June* 11, 1896.

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21. *Malignant endocarditis in an infant.*

Exhibited by JOSEPH SAILER, M.D.

The subject was born normally, and continued well until fourteen days old. At that time she refused to take nourishment, and died in two days without fever having been noticed.

The body was that of a well-nourished female child; no external marks, and the umbilicus in particular was perfectly normal. There

was a slight mucopurulent discharge from the nostrils. The peritoneum was normal. The spleen slightly enlarged and very soft; the liver and kidneys showed a slightly cloudy section; the lungs were congested. The heart was of normal size; the pericardium normal. The tricuspid valve was slightly thickened; the pulmonic valves normal. The posterior mitral valve had a few red prominences upon its free surface that were firmly adherent to the endocardium, both leaflets had slight thickenings of the endocardial lining; the aortic valves were normal. The pathologic diagnosis was endocarditis verrucosa acuta et septicæmia.

The diagnosis is made upon the following grounds: 1, there is no anomaly in the formation of the heart; 2, the predominance of the lesions of the left side; 3, the nature of the lesions, being probably mycotic thrombi into the valve; 4, the history of the case, the patient being apparently healthy until within two days of death; 5, the presence of a rhinitis that might have served as the entering point for the germs.

Vegetative endocarditis of the infant not consecutive to fetal endocarditis is rare, but a few cases have been reported (André, Petit), and the present case seemed to be of sufficient importance to justify its presentation.

Discussion.—Dr. Riesman said, with regard to heart disease being associated with tuberculosis, it was generally held that a strong antagonism existed between the two conditions. It had been claimed that the excess of blood-serum in the congested organs acted as a bactericidal agent. This view had led to the treatment of tuberculosis of joints by passive congestion, and it had even been advanced that pulmonary tuberculosis should be treated in this way. *June* 11, 1896.

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22. *Aneurysm of the abdominal aorta, with thrombosis of the right renal artery.*

Exhibited by J. H. Musser, M.D., and J. Dutton Steele, M.D.

The patient was a negro, aged forty-seven years, a native of New Jersey, and a laborer by occupation. He was admitted to the medical wards of the Philadelphia Hospital, May 1, 1896. His mental condition was such that the following data were obtained with difficulty:

His mother, father, and five brothers are dead of unknown causes.

Four brothers and one sister are alive and healthy. There is a history of alcoholism in the family, but no tuberculosis or rheumatism. He had had typhoid fever and pneumonia in boyhood, but never scarlet fever. No history of syphilis or alcoholism could be obtained. For two weeks previous to admission he had had much pain in the left iliac region, running across to the right hypochondriac. He was constipated, and had had several obstinate attacks of hiccough. There is no history of ascites or edema at any time. The patient is delirious at intervals, and his statements are indefinite and unreliable.

Examination, upon admission, by Dr. Musser: There is considerable emaciation and muscular wasting. The pupils are contracted and are equal. The patient is in a condition of muttering delirium and can be aroused with difficulty. The abdomen is contracted to a marked degree, and the muscles of the abdominal wall are so rigid and there is such exquisite tenderness over this entire region that no data can be obtained by the usual methods.

Superficial cardiac dulness extends three and a half inches to the left of the midsternal line, and by auscultatory percussion three-fourths inch farther to the left. The right border corresponds with the mid-sternum. The upper limit is the upper border of the third rib. The apex-beat is in the fifth interspace in the midclavicular line. The heart-sounds at the apex are muffled and indistinct. The second sound in the aortic area is accentuated and bell-like in character. The pulse is of high tension, and the radial and temporal arteries are thickened and rigid.

There is an area between the right nipple and the right edge of the sternum where the resonance on percussion is high-pitched, the tactile fremitus is lessened, and the breath sounds are suppressed. The heart-sounds are transmitted to and clearly heard in this region. Vocal resonance is increased over the apex of the right lung anteriorly, but the breath-sounds are suppressed over both apices. Posteriorly there is suppression of the respiratory murmur over the upper third of both lungs. It is fairly distinct at the bases. There are no râles.

The upper border of the liver dulness corresponds with the upper border of the seventh rib.

Urine. The amount secreted in the twenty-four hours following admission was twenty-four ounces. Its specific gravity was 1020. It was acid in reaction, and contained one-fourth, by bulk, of albumin, many pale granular casts, and much uric acid.

The temperature upon admission was 99° F., but it soon became subnormal, and ranged between 96° and 97° F. until death.

The amount of urine increased slowly to fifty-two ounces in the twenty-four hours upon May 12, and then gradually diminished in amount until death. Its chemical analysis remained practically the same.

The patient had several severe and obstinate atttacks of hiccough while under observation. He was constipated, and vomited frequently. The mental symptoms grew progressively worse, and five days before death he passed into a condition of stupor. Death occurred May 17.

Autopsy (at the Philadelphia Hospital, May 18, thirty hours after death). The body of an emaciated negro. The abdomen is markedly retracted over its whole extent. The muscles of the abdominal wall are firmly contracted, and on section are of a dark, slaty-red color (postmortem change?). There is very little subcutaneous fat. Rigor mortis is well marked.

Abdominal cavity. There is a small amount of clear fluid. No evidence of tuberculosis upon any of the peritoneal surfaces. The liver reaches to the fifth interspace above, and extends barely to the costal margin in the midclavicular line. Behind the pancreas, and lying upon the body of the first lumbar vertebra, is a rounded tumor. This is retroperitoneal, and is opposite the origin of the seventh costal cartilage. The inferior vena cava and the nerves of the solar plexus lie upon its anterior surface. This tumor measures six centimeters in its anteroposterior diameter and eight centimeters in its vertical diameter. On dissection, it is found to be a saccular aneurysm of the abdominal aorta. The celiac axis is given off immediately above the sac, and is adherent to it. The superior mesenteric artery comes from the aorta immediately below it. The left renal artery arises from the aorta directly behind the opening of the sac, and the right renal artery is given off from the sac itself. Upon opening the aorta, the mouth of the dilatation is found to be clearly defined, and measures three by two centimeters. The long diameter of the opening is in the same direction as that of the artery. The sac is entirely filled with a red and firm coagulum. This has a definite, laminated structure, which is especially marked in the portion in relation with the aneurysmal wall.

When the right renal artery is opened, it is seen to contain a hard, white thrombus that appears totally to occlude the artery for a distance of eighteen millimeters from its point of exit from the aneurysm. This thrombus mass is closely adherent to the arterial wall, from which it can be stripped with difficulty. It is firm and resisting, and has evidently undergone fibrous change. The artery and its branches to the distal side of the thrombus are filled with softer red coagula, until the

kidney substance is reached, when the coagula cease, and the vessels are free from obstruction. The left renal artery is normal. The intima of the abdominal and thoracic aorta shows many areas of fibrous change.

The connective-tissue envelope of the aorta was stripped off down to the aneurysm, and at one portion of the sac was found to constitute its only covering. At other points the coats of the artery appear to be intact. The inferior vena cava lies directly to the right of the tumor and receives the left renal vein that crosses the tumor to join it, and is much dilated, measuring two centimeters in diameter at its widest point. The vein of the right kidney is normal.

Pleural sacs. The right is almost entirely obliterated by firm adhesions. The left has a few adhesions posteriorly at the apex.

Lungs. Hypostatic congestion and edema of the lower lobes on both sides. Otherwise nothing of interest.

Pericardium. Normal.

Heart. Full of liquid blood. The left ventricle is firmly contracted. Its myocardium is of fairly good texture and is much hypertrophied. The wall of the left ventricle measures twenty-four millimeters in thickness. There is considerable atheroma of the aorta directly above the semilunar valves, but the leaflets themselves are not affected. There is slight fibrous thickening of the free edges of the mitral leaflets, but the valve appears competent. The right ventricle is dilated and its myocardium is somewhat hypertrophied. It measures six millimeters in thickness. Valves of the right heart are normal. The weight of the heart is 440 grams.

Spleen. Small and is much congested. There is evidence of considerable increase in connective tissue. Its weight is 60 grams. Dimensions, 9 x 7 x 1 centimeter.

Suprarenal capsules. Normal.

Left kidney. Somewhat enlarged and measures 12.5 x 9 x 3 centimeters. The capsule strips readily. The organ is somewhat flabby. The stellate veins are plainly seen. The cortex is swollen, and is generally of a pale-yellow color. The pyramids and medullary rays are congested.

Right kidney. Is smaller than usual, and measures 11 x 6.5 x 3 centimeters. It is intensely congested, and drips blood from the section. The capsule is somewhat adherent, and there are several small cysts upon the surface under it. The cortex is contracted and its consistency is increased. The cortex and pyramids are uniformly congested and dark-red in color.

Ureters and bladder. Normal.

Pancreas. Normal.

Small intestine. Normal. No evidence of tuberculosis.

Stomach and duodenum. Nothing of interest is noted.

Colon and rectum. There is a large amount of feces in scybalous masses. Both show irregular areas of intense congestion. The mucous membrane is covered by tenacious mucus. In the rectum are numerous hemorrhages into the mucous membrane, which average two centimeters in diameter. The impression is given that such areas are the result of pressure caused by the fecal masses.

Liver. Somewhat pale and irregularly mottled by yellowish areas. On section there is seen to be considerable perilobular congestion, and the parenchyma is pale, with scattered yellow spots that appear fatty.

Gall-ducts are patulous and are not dilated.

Gall-bladder. Normal.

Brain, spinal cord, and their membranes. Normal.

Microscopic examination: Left kidney. There is some increase of the connective-tissue elements, as well as evidences of an acute inflammatory process. The glomeruli, as a rule, are swollen, the capillaries are dilated, and the connective tissue of the tufts shows a considerable amount of round-cell infiltration. In places the tufts have undergone a hyaline degeneration that appears to commence in the capillary walls. The Malpighian bodies become swollen and homogeneous with fragmentation, and occasionally complete loss of the nuclei. The hyaline material stains poorly with carmin, and when the process is not too far advanced the faint outlines of the individual capillaries can still be seen. The cells of the tubules are much swollen throughout the cortex. In places there are much desquamation and an advanced fatty degeneration. The arterioles throughout the section show much increase of the connective tissue of the intima and adventitia. In many instances the endarteritic process has caused complete obliteration of the lumen of the vessel. Occasionally commencing hyaline degeneration is seen in such an obliterated arteriole. There are numerous small hemorrhages scattered through the cortex.

Right kidney. There is some increase in the connective tissue. This condition is rather more marked than in the left kidney. The Malpighian bodies are swollen and infiltrated, and in several places there is commencing hyaline degeneration, similar to that described above. The cells of the convoluted tubules have very generally undergone fatty degeneration, and in many instances have lost their nuclei and have entirely broken down, the tubules being filled by granular detritus.

The arterioles show an endarteritis, with occasional obliteration of the smaller vessels, and commencing hyaline change. There are no hemorrhages in this kidney.

This case has seemed to us to be of interest, both on account of the rarity of thrombosis of the renal artery and the peculiarity of the clinical symptoms.

There was no attempt, macroscopically at least, at collateral circulation between the branches of the occluded renal artery and the small branches of the lumbar vessels supplying the perirenal connective tissue. Indeed, in the authorities that we have consulted there is no mention of such an anastomosis, but from the close proximity of the terminal branches of the two sets of vessels such a communication is conceivable. The only apparent results of the stoppage of the arterial supply were the intense congestion of the organ and the advanced degeneration of the cells of the convoluted tubules. It is remarkable that the left kidney maintained the urinary secretion as long as it did, in view of its diseased condition and the evident inability of its fellow to assist it.

The previous history, that of the present disease, and the subjective symptoms did not aid us in the diagnosis. That of nephritis and uremia was established by the results of the urinary examination, in connection with the cerebral and gastric symptoms, which were looked upon as uremic.

Of great interest and difficult to explain were the markedly hard and retracted abdominal walls. The emaciation of the subject led to the suspicion of possible tubercular peritonitis or gastric carcinoma. Such remarkable retraction is seldom seen except as the result of peritoneal irritation in an aged subject. It is known to occur, of course, in meningitis of childhood.

The great amount of sclerosis throughout the whole arterial system may perhaps be explained by alcoholism, though the extensive endarteritis of the smaller vessels of the kidney suggests a possible syphilitic origin. *June* 11, 1896.

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23. *Tricuspid, mitral, and aortic stenosis.*

Exhibited by J. Dutton Steele, M.D.

This specimen was placed at my disposal by my colleague, Dr. Sailer, who performed the autopsy.

Olga S., aged thirty-nine years, a native of America; single, a teacher

by profession. Father was insane, and died of phthisis. Mother died at thirty-two years of cholera. One uncle was insane. The patient contracted acute articular rheumatism in 1890, and was bedridden from this attack for three years. She had had a slight attack of rheumatism in early life. In 1893 she developed symptoms of paranoia and was committed to the insane department of the Philadelphia Hospital, where she remained until her death, on June 15, 1896. Cardiac symptoms seem to date from the rheumatism acquired in 1890. She had repeated attacks of loss of compensation, one of which proved fatal.

Autopsy (June 16, 1896). Abdomen, pleura, and pericardium contained large quantities of clear blood-stained fluid. In the latter there were also a few old adhesions.

Heart. Left ventricle 10 centimeters long and 6 broad. Right ventricle same dimensions. Right auricle tremendously dilated. Tricuspid orifice does not admit the tip of little finger. Upon the anterior surface are a few marantic clots firmly adherent to the auricular wall. The three leaflets of the valve are united, leaving a triangular orifice 7 by 5 millimeters. The three leaflets thus united form a funnel-shaped sac with the orifice at the bottom projecting into right ventricle. The edges of the valve are studded with small recent vegetations. The wall of the right auricle measures 3 millimeters in thickness; right ventricle 7 millimeters. The pulmonic leaflets show sclerotic bases, and are slightly adherent at their edges. Intima of pulmonary artery is much congested. The left ventricle is greatly dilated. Mitral valve is buttonholed, and does not admit the tip of little finger. The upper surface of the valve is rough and shows a few minute vegetations. Left auricle 3 millimeters in thickness; left ventricle 15 millimeters. Aortic valve agglutinated by adhesions of the leaflets into a dense, sclerotic ring, forming a narrow slit 15 millimeters in circumference, into which the tip of the little finger cannot be passed.

There are a few patches of atheroma just above the attachments of the aortic valve. Myocardium dense and sclerotic. Coronary arteries are slightly sclerotic.

Lungs show acute emphysema, much edema, and hypostatic congestion. Spleen is seat of cyanotic induration. Kidneys show chronic interstitial inflammation, and a pyramidal scar, with base toward periphery. Stomach reveals chronic inflammation and fibrous stenosis of pylorus. The brain is apparently normal.

It does not seem to me that the changes in the heart are the result of a fetal endocarditis, for the following reasons:

1. The age of the patient.

2. The fact that she was of good development, and not under-sized, as occurs in so many cases of congenital heart-disease.

3. The pericardial adhesions suggest the occurrence of inflammation around the heart at some period.

4. The pyramidal scar in the kidney is probably the remains of an infarct that might have readily resulted from endocarditis in adult life.

5. The absence of any defect in the heart's development.

6. The fact that the cardiac symptoms, if they did not originate in the attack of rheumatism mentioned, certainly were much more severe after its occurrence.

Lesions of the right side of the heart, acquired in postnatal life, can no longer be considered extremely rare. Sansom, in his *Diseases of the Heart and Aorta*, states that in his experience the right side of the heart is affected in quite 50 per cent. of all acute inflammations of the endocardium, and in almost every case of severe endopericarditis. This statement is supported by Byron Bramwell in his article in the *American Journal of the Medical Sciences*, published in 1886. They both state that the right heart is subjected, however, to so much less strain than the left, that resolution much oftener takes place, and so there is less likelihood of the formation of permanent lesions. In the present case, whatever was the time of occurrence of the attack of endocarditis giving rise to such extensive changes, an explanation may be sought for the fact that the acute inflammation existing at the time of death is more marked on the right side than on the left. The reason seems evident. The extreme difficulty of getting blood through the initial orifice must have thrown tremendous strain upon the right heart, and the amount of work demanded by the respective sides must have been the reverse of that required in health. Therefore the right side became the point of less resistance and more liable to infection. This specimen is more interesting in the degree of stenosis than in the number of valves affected. In eighty-six cases collected from literature in which the right heart was affected, 30 or 35 per cent. showed disease of the tricuspid, mitral, and aortic valves. In seven of these there were pericardial adhesions. This proportion is rather larger than Osler gives, which is 18 per cent. in 114 cases. In the series mentioned the greatest degree of stenosis occurs in a case reported by Simpson in the *British Medical Journal* for 1868, where the tricuspid valve admitted one finger to the first joint and the mitral measured 6 by 3 millimeters. All authorities unite in the statement that the right heart is diseased much oftener in women than in men.

Discussion.—Dr. Allyn said: According to Broadbent, tricuspid stenosis was the most frequent accompanying lesion of mitral stenosis. It was oftentimes difficult to make this out unless there was some difference in the pitch or duration of the murmurs. Dr. Steele had referred to the fact that this case occurred in a woman, and he was especially interested in it on that account, as he had looked up the matter in connection with pregnancy. One-half the cases ended fatally in the first or some subsequent pregnancy.

Dr. Kelly, referring to Dr. Allyn's comments, said that he did not hear whom Dr. Allyn had quoted as stating that tricuspid stenosis was the most frequent accompaniment of mitral stenosis, but he certainly thought this a mistake, as he did not believe it was so. As to the murmur in connection with tricuspid stenosis, he thought it had very little to do with the diagnosis. *June* 25, 1896.

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24. *Tuberculous pericarditis.*

Exhibited by A. A. Eshner, M.D., and J. D. Steele, M.D.

Autopsy. The body of a somewhat emaciated man. Pericardium is completely obliterated. The layers are much thickened, measuring, together with exudate between them, 4 millimeters. The surface of the two layers, when stripped apart, are seen to be studded with small, round, opaque bodies about 1 millimeter in diameter, and of a whitish-gray color. These bodies are also scattered through the connective tissue between the layers of the pericardium. The adhesions are so dense that in many places the layers cannot be separated.

Heart. Muscle about normal in thickness. In the myocardium of the left ventricle there is much over-growth of connective tissue. Myocardium of right ventricle is friable, but shows no sclerosis. The aorta is intensely atheromatous. The aortic leaflets are slightly adherent. The mitral orifice is somewhat narrowed. The posterior leaflet is much thickened and is calcareous. The orifice admits three fingers to the first joint, and appears incompetent. The posterior leaflet of the tricuspid valve shows a few fibrous nodules along the line of contact. The pulmonary valves are normal.

Pleura. Some adhesions on both sides posteriorly, and both sacs are three-quarters full of clear, serous fluid. The posterior mediastinal glands lying in close contact with the pericardium are generally

enlarged, and several show extensive cheesy degeneration in their centres.

Lungs. Several old scars at each apex containing a few chalky masses that are evidently tuberculous.

Kidneys and liver are cyanotic and show interstitial change.

Microscopic examination of pericardium. The round masses described present the typical appearances of miliary tubercles. The connective tissue is infiltrated with round cells. In no place is degenerative change noticed, and the tuberculous process appears to be acute.

While tuberculous inflammation of serous membranes may form dense fibrous adhesions, there is usually considerable cheesy degeneration accompanying such change. The density of the fibrous bands between the layers, and the absence of any caseous change, either in the tubercles or adhesions, suggest the possibility that the sequence of events in this case may have been first an obliterating pericarditis, nontuberculous in character, occurring simultaneously with the endocarditis that caused the aortic and mitral lesions, and then a secondary infection by tubercle bacilli from the mediastinal glands.

Discussion.—Dr. Sailer said Dr. Steele had given very good reasons why this should be called "Obliterating Pericarditis," and had almost convinced him that he was right. Serofibrinous pericarditis is usually due to some tuberculous origin, and it seemed to him that this might be called tuberculous.

The President said he thought it would be fair to call Dr. Steele's case of "Obliterating Pericarditis" one of "Tuberculous Pericarditis."

June 25, 1896.

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25. *Thrombosis of the pulmonary artery.*

Exhibited by Dr. A. E. Taylor.

The specimens which I herewith present, together with a drawing, were taken from the body of a child of ten days. The child was born by normal labor, and seemed healthy at birth. The cord was discarded in the normal time and manner. On the eighth day the child was attacked with fever, dyspnea, and rapid cardiac excitement. Although no rigors could be made out, a diagnosis of pneumonia was made. The condition rapidly became worse, and in two days the child died in extreme cyanosis. The autopsy was done two hours after death. The

cord was healthy. There was a complete occluding thrombosis of the mouth of the pulmonary artery; the lungs were not consolidated, but were hard, and presented numerous hemorrhages. The heart, its valves and covering were normal. There were hemorrhages in the lymph-glands, liver, spleen, pancreas, kidneys, and nervous system. Microscopically, in all the tissues named there was a marked fibrosis, with proliferative periarteritis and interstitial hemorrhages, accompanied by parenchymatous degeneration of slight degree. I stained over a hundred sections of the thrombosis. It was a simple fibrinous formation, and bacteria were not to be found, nor were they to be found in the other tissues. I believe that the absence of micro-organisms and the presence of the connective tissue and arterial overgrowth make the diagnosis of congenital syphilis very probable. Quite similar cases, apart from the pulmonary thrombosis, have been described as syphilis neonatorum hæmorrhagica. The condition of a thrombosis of the root of the pulmonary artery is very rare. I have not had the opportunity of reviewing the entire literature. As yet I know of but two such cases reported. *September* 24, 1896.

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26. *Multiple aneurysms of the aorta.*

Exhibited by Dr. Augustus A. Eshner.

R. S., a decrepit German, seventy-four years old, who had formerly been a florist, was admitted to the medical wards of the Philadelphia Hospital on June 2, 1896, presenting no especial features beyond those referable to his years. He himself attributed his condition to the excessive use of tobacco. The action of the heart was arhythmic, but no murmur could be heard. The vessels generally were extremely atheromatous. On admission the temperature was normal, but on July 7, when the patient remained in bed on account of not feeling well, the thermometer was found to register 102.4°. The temperature gradually subsided, while the patient grew progressively weaker, complaining especially of pain referred to the lumbar region. Death took place on July 27, amid manifestations of general asthenia.

The postmortem examination was made by Dr. Jos. Sailer, from whose report these notes are taken.

On raising the small intestines a large retroperitoneal hemorrhage was discovered, occupying chiefly the left side of the abdominal cavity. The blood had infiltrated all of the retroperitoneal structures. The

clot was soft and dark-red in color, and on pressure serum escaped. A huge mass of blood had infiltrated the perirenal fat. The abdominal aorta appeared greatly distended. The splenic artery was exceedingly tortuous and atheromatous, as were all of the other arteries of the abdominal cavity.

The thoracic aorta was moderately dilated just beyond the vessels arising from the arch, and the vessel was large and tortuous.

The glands of the superior mediastinum were large, black, firm, and edematous.

The pericardium contained only the usual amount of fluid.

The heart was of about normal size. Its vessels were moderately tortuous. There was a considerable amount of pericardial fat and some mottling of the posterior surface from the presence of whitish patches. The bicuspid orifice admitted four fingers. The valve-leaflets were soft in consistency, though they presented a moderate amount of thickening, with a small patch of atheroma in the endocardium just above the posterior leaflet. The wall of the right ventricle was soft and the muscular structure friable; it was 7 mm. thick. The endocardium of the left ventricle presented numerous irregular, whitish striæ. The bases of the mitral leaflets were infiltrated with atheroma. The chordæ tendineæ were long and flabby. The papillary muscles were yellow, fibrous, and considerably reduced in size, and contained calcareous nodules. The mitral orifice admitted two fingers. The edges of the valve-leaflets were moderately thickened. The myocardium of the left ventricle was pale, friable, and soft, and contained numerous patches; it measured 20 mm. in thickness at the base. The aortic valves were sclerotic, containing on the posterior surface calcareous nodules along the line of contact. The base of the aorta showed moderate infiltration. The coronary arteries were transformed into rigid, calcareous tubes.

The lungs were emphysematous and edematous. The upper lobes contained numerous areas of infiltration, with caseous and calcareous degeneration at the center.

The spleen was large and soft. Its capsule was the seat of a small patch of firmly adherent fibrin. On section the splenic tissue presented a mottled appearance, and contained a few hemorrhages. The splenic artery throughout its whole course was exceedingly tortuous and rigid.

The kidneys were contracted and granular, containing also a few cysts.

The bladder was contracted, its walls thick.

The stomach was of normal size. Its mucous membrane was thick-

ened, rough, and rugous, and contained a number of punctiform hemorrhages.

The pancreas appeared to be normal, except for some interlobular hemorrhage. The capsule was slightly thickened, and contained a number of whitish spots.

Just beyond the origin of the vessels arising from the arch of the aorta, there was a dilatation about five cm. in diameter. The walls of this dilatation were dense and atheromatous, and were torn in manipulation. There was, however, no extravasation of blood or other evidence of rupture during life. The whole of the arch and the descending aorta showed atheromatous infiltration, with numerous areas where the intima had been destroyed by ulceration. In the abdomen the celiac axis was considerably dilated and its branches tortuous and rigid. Below the axis the renal arteries emerged, and just below this point there was a large sacculated dilatation of the aorta extending downward, just above the bifurcation. This sac would easily contain a closed fist, and was lined with laminated clots, some of which could be seen from the external surface. At the upper anterior aspect of this dilatation was a large ragged opening communicating with the retroperitoneal space. *September* 21, 1896.

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27. *Aneurysm of the right posterior aortic sinus of Valsalva, with rupture.*

Exhibited by Henry W. Cattell, A.M., M.D., and J. Dutton Steele, M.D.

Aneurysm of one of the sinuses of Valsalva is a very rare pathologic lesion, as indicated by the scant references to this condition in medical literature, and by the fact that this is the first case of the kind which one of us has seen in some two thousand autopsies.

Our case is that of a well-developed colored man who was found dead October 9, 1896, his age being about forty-five years. On the frenum of the penis is a healed scar, probably of syphilitic origin. The abdominal cavity contains about two quarts of clear fluid. The peritoneal covering of the intestines is normal. The liver barely reaches to the costal margin in the midclavicular line. There is considerable perihepatitis. The omentum is shortened and thickened, and there appears to be a fibrous change in it. It is rolled up and contracted, tightly adherent to the transverse colon, and forms a mass 5 cm. in vertical and

4 cm. in antero-posterior diameter. The parietal peritoneum is thickened, white, and glistening. Mesenteric glands are somewhat enlarged. Appendix is free, but shorter than normal. The left pleural cavity is half full of clear yellow fluid, evidently transudatory; no fibrin in it. No evidence of an inflammatory process. Right pleura is about one-third full of a similar fluid. No adhesion in either pleura. Pericardium contains about 100 c.c. of clear fluid. Several milk-spots are seen in pericardium on anterior surface of heart. Heart is dilated and relaxed, and left ventricle is full of liquid blood. The auricles and left ventricle contain firm white clots. The coronary arteries are tortuous and show many plates of fibroid change. The heart is much hypertrophied, and weighs twenty ounces (566 grams). The muscle is friable, and there is some evidence of fatty and fibroid changes in the papillary muscles; the mitral valves are slightly thickened and opaque. The aortic valves show several spots of commencing fibroid change; the valves of the right side are normal. There is an aneurysm of the right posterior sinus of Valsalva (the one in which there is no coronary artery). This aneurysm admits two fingers and affects this sinus alone, the other two aortic leaflets not being disturbed. The aneurysm extends downward and backward to the right and pouches into the right auricle. It has dissected up the endocardium of the auricle, and has dissected loose the attachment of the posterior tricuspid leaflet and the end covering the auriculoventricular ring. The posterior lining of the aneurysm is formed by the dilated wall of the aorta, which has undergone much fibroid change, and in several places shows calcareous plates. The apex of the aneurysm just above the attachment of the posterior tricuspid leaflet shows a small opening just admitting a small probe, leading from the aneurysmal sac into the right ventricle. The opening is surrounded by a ring of soft, newly-formed granulation-tissue. The aneurysmal pouch measures, in width, 4 cm., anteroposterior diameter, 3.7 cm.; the hole is 12 mm. from the free edge of the tricuspid valve. Circumference median, open 8.5, when shut 6 cm.; depth 3 cm. The left ventricle is 20 mm. thick; left auricle is 5 mm. thick; right ventricle is 10 mm. thick; right auricle is 4 mm. thick.

Spleen: Capsule is much thickened, opaque, and white. There is considerable perisplenitis binding the organ to the abdominal wall and to the transverse colon. There are no scars upon the organ. Dimensions, 8.5 x 7.5 x 2.5 cm.

Right kidney is somewhat tense and hard. Capsule strips well, but there are several small cysts under it in the cortex. The organ is intensely congested, but the cortex is not contracted. The organ is seen

to be in a condition of cyanotic induration. Malpighian bodies very prominent. Measurements, 10 x 2.5 x 8.5 cm.

Left kidney practically shows the same condition, but no cysts; upon the under surface of the organ is a depressed scar that is puckered and stellate. It is superficial, and does not extend into the body of the organ. It is white and glistening. It measures 10 x 5.5 x 2.5 cm. Suprarenal much congested. Bladder and ureters normal. Pancreas normal. Intestines normal. Stomach normal.

Liver: Edges rounded; peritoneal coat over whole organ is opaque, white, glistening, and measures 2 mm. in thickness. The gall-bladder is likewise thickened. No scars extending beneath the surface. On section the organ is of a diffuse yellow-orange color, alternating with deeply congested areas around the acini, which also are deeply congested. The acini are plainly marked, and there is evidently an increase of connective tissue. Bile-ducts are not dilated, and at the hilum seem normal.

Brain and membranes normal; skull somewhat thinner than usual, but no areas of necrosis.

Lungs show no scars upon the pleural surface. Several bronchial glands show extensive softening in their centres.

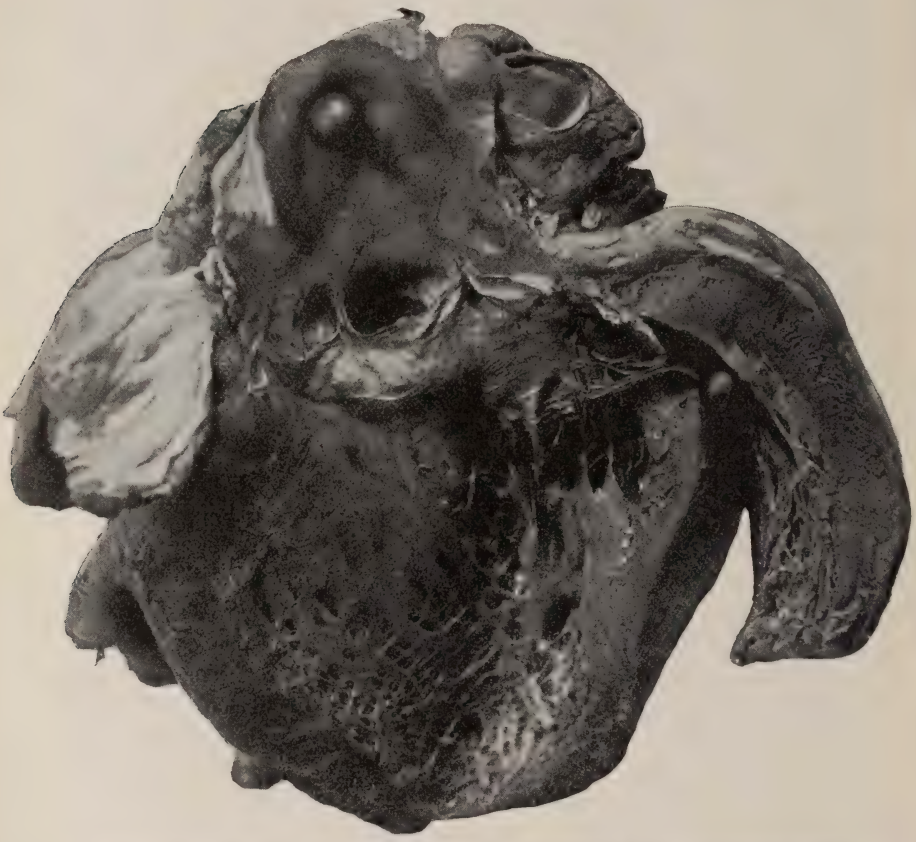
Microscopic examination revealed a slight increase in the connective-tissue elements throughout the different organs, but nothing of interest besides this. The scar in the left kidney showed nothing characteristic. There was a moderate amount of endarteritis in both kidneys.

The clinical history of the case being unknown to us, it would seem probable from the postmortem findings that the aneurysm was due to a syphilitic change starting in the walls of the sinus of Valsalva. The rupture probably took place some time previous to death, and would have caused an admixture of the arterial with the venous blood. The cause of death seems to have been fatty degeneration of the myocardium complicated by the aneurysm and sclerotic changes in the parenchymatous organs.

In the seventeen volumes of the TRANSACTIONS OF THE PHILADELPHIA PATHOLOGICAL SOCIETY there are reported 101 aneurysms, of which 62 were of the arch of the aorta and but 2 of the sinus of Valsalva. Bosdorff reports 48 aneurysms (exclusive of the miliary forms) in a series of 3108 autopsies: 28 were situated in the arch of the aorta and 2 were in the sinus of Valsalva. In the last twelve volumes of the *Middlesex Hospital Reports* there are records of 3030 autopsies. Among these are 45 aneurysms (exclusive of the miliary cerebral forms): 30 occurred in the arch of the aorta and 2 were of the sinus of Valsalva.

Combining the figures just given, there are 120 aneurysms of the aortic arch and 6 of the sinus of Valsalva.

FIG. 1.



Dilatation and bulging of the right aortic sinus of Valsalva are seen; the coronary artery on the left side comes off above and on the right side below the free margin of the valve. The aortic and mitral valves are smooth, while some fibrous change is noticed in the tips of the papillary muscles. (Reduced one-third.)

In the total number of aneurysms of the list given by Bosdorff and the *Middlesex Hospital Reports* (103) the percentage of this condition was 3.8 per cent. Perhaps some of these cases were nothing but dilatations which should not be counted as true aneurysms, a condition which we have seen in at least three cases.

In 1840 Thurnam collected 22 cases of aneurysm affecting these aortic

sinuses, which were distributed as follows: 12 in the right sinus, 4 in the left sinus, and 6 in the posterior sinus.

FIG. 2.

The aneurysmal bulging of the left auricular wall is seen presenting in the tricuspid orifice; the leaflets of the tricuspid valve have been separated, and therefore appear distorted. The probe passes from the right aortic sinus through the point of rupture in the aneurysm. The latter would therefore discharge blood into the right ventricle, even if there had been complete closure of the valve, as the pulmonary cusp of the tricuspid at its side is composed of chordæ tendineæ. (The photograph has been taken from above looking down upon and through the tricuspid orifice; the pulmonary artery is at the top and to the left. Reduced one-third.)

Of the aneurysms in the right sinus 2 had ruptured into the pericardium and 1 into the right ventricle; 2 projected into the pericardium and 1 into the right ventricle; 1 projected into the left auricle and 1 into the left ventricle. Of the 6 in the posterior sinus 2 had perforated,

1 each into the right and the left auricle. From the character of the opening in the aneurysms that ruptured back into the circulation, comprising three of his list, he concludes that the opening had been present for some time before death, and calls the condition a spontaneous varicose aneurysm. Our case belongs to this class, as do the majority of those that we have collected from literature since Thurnam's work. The rupture, of course, does not lead to immediately fatal results, and death apparently occurs oftenest, as in our case, from fatty degeneration of the hypertrophied myocardium.

Sibbs has collected 860 cases of aneurysm. Of these 632 were of the arch of the aorta and 87 involved the sinuses of Valsalva, or 13.8 per cent.

We have collected 21 cases from the literature on this subject, in a number of which the antemortem condition is noted.

Ogle: Male, aged forty-two years. Aneurysm from posterior sinus of Valsalva, projecting and rupturing into the upper and inner parts of the right ventricle. The opening from the sac was plugged with a firm coagulum. Distinct systolic murmur heard at apex. Heart much enlarged and dilated.

Fischel: Aneurysm of one of the aortic sinuses rupturing into left ventricle.

Gage: Male, aged nineteen years. Aneurysm from one of the sinuses, admitting phalanges of five fingers, projecting and rupturing into right ventricle. Much hypertrophy and dilatation of heart, especially affecting the right ventricle. A double murmur in the aortic area, the systolic transmitted up and the diastolic down.

Lichtenberg: Male, aged twenty-nine years. Aneurysm of posterior sinus, with an opening into the right ventricle between the anterior and posterior tricuspid leaflets. The sac is the size of a filbert. A double murmur at the base of the heart and in the pulmonary area.

Jansen: Aneurysm of one of the sinuses, projecting into the right auricle, without rupture. Aortic valves extensively diseased.

Pepper: Aneurysm of one of the sinuses, projecting into the right ventricle by dissecting the muscle and endocardium. The opening of the sac into the aorta was one and one-half inches in diameter. It had ruptured into the pulmonary artery, immediately above the leaflet, by an opening half an inch in diameter. The aortic valves are healthy. The heart is much enlarged, and the left ventricle is especially hypertrophied.

Stokes: Male, aged thirty-one years. Aneurysm of one of the sinuses, projecting and rupturing into the conus arteriosus pulmonalis. Slight dyspnea and much edema of legs and scrotum. A thrill was felt over

the sternum, and a double murmur was heard, best at the base, but loud enough to be heard all over the chest and also by the patient himself. The systolic murmur was splashing in character and not transmitted to the carotids. Some days before death the patient experienced a sharp pain in his chest, and became weaker. The thrill was henceforth felt only over the apex, and the double murmur was heard best at that point.

Bosdorff: Male, aged fifty-three years. Aneurysm of one of the sinuses of Valsalva. Clinical diagnosis: Endocarditis and central embolism. There was hypertrophy of the heart, with fatty degeneration and numerous old and recent thrombi in the brain. Atheroma of the aorta and basilar artery.

Bosdorff: Male, aged thirty-six years. Aneurysm of one of the sinuses of Valsalva. Clinical diagnosis: Endocarditis and insufficiency of aortic valves. There were also cardiac hypertrophy and fatty degeneration, and thrombosis of coronary artery.

F. P. Henry: Male. Aneurysm of the right sinus of Valsalva, which has opened into the pulmonary artery. The opening is at the point of union of two of the leaflets of the pulmonary valve. Physical signs: Dulness in the left intraclavicular region. Pulsation and thrill in the second intercostal space to the left of the sternum. A rasping systolic murmur heard best in the same area. No pressure symptoms. Much edema of the legs and scrotum.

Middlesex Hospital Reports: Male, aged thirty-four years. Aneurysm of the left sinus of Valsalva, opening into the conus arteriosus of the pulmonary artery, just below the pulmonary valves. There was a history of alcohol to excess, but not of syphilis, and severe pain on exertion three weeks before death.

Middlesex Hospital Reports: A saccular aneurysm of the right sinus of Valsalva, about the size of a racquet-ball, projecting into the pericardium. There was no edema. The aortic valves were incompetent and much thickened. The heart was dilated. There was a fusiform aneurysm in the descending portion of the arch of the aorta.

Openchovsky: In this case the aneurysm was large and followed by disease of the valves of the heart. The article is illustrated.

Beyer: Male, aged thirty-three years. Aneurysm of the right anterior sinus of Valsalva, the size of a walnut, one inch below the pulmonary valves. The left anterior sinus also dilated, but not to the extent that the right anterior showed. This sac had pushed its way in between the pulmonary artery and the aorta, and was about to rupture into the pericardium. There was a history of rheumatism and syphilis. Physical signs were a double murmur in both the mitral and aortic

areas. The heart was fatty. Microscopic sections of the various organs showed a general slight hyperplasia of the connective tissue.

Hawkins: Male, aged forty-six years. Alcoholic history. Died suddenly, after exertion. No physical examination. Aneurysm of the anterior sinus of Valsalva about the size of a filbert. The opening of the right coronary artery is at the base of this aneurysm. There is another aneurysmal sac involving the left posterior sinus and the wall of the aorta above the valve. The sac was the size of a hazelnut. There is still another dilatation opening from the aorta above the right posterior sinus, which is the size of a hen's egg and had ruptured into the pericardial sac.

Osler has seen ulceration in one of the sinuses of Valsalva which extended into the septum. The upper part presented an aneurysmal dilatation which had ruptured into the left ventricle.

Foster: An aneurysm of one of the aortic segments, which caused during life a soft systolic murmur over the valves, the sac finally rupturing.

Durand-Fardel: Female, aged forty-nine years. No rheumatic history; alcoholic. For six months she had attacks of suffocation and palpitation of heart, accompanied by swelling of the lower limbs. In one of the attacks her face became congested, lower limbs edematous, with extreme oppression and anxious countenance. Lungs filled with moist râles; heart-action increased and irregular; no murmur; pulse filiform; urine scanty and containing albumin. Three days after the subsidence of the attack a distinct murmur of aortic insufficiency developed, without the characteristic pulse. Digitalis had no action. Her condition gradually grew worse, the heart murmur remaining constant.

The heart was considerably hypertrophied, weighing 500 grams. The aortic leaflets were atheromatous and studded with calcified nodules. Behind the posterior leaflet was an aneurysm the size of an English walnut, with a rounded orifice two centimeters in diameter. Its walls were calcified in places. It extended in the direction of the interauricular wall. The coronary arteries had no connection with the sac. No general arterio-capillary sclerosis.

Van Iterson: Aneurysm, the size of an egg, of the right sinus of Valsalva, which bulged toward and finally ruptured into the left ventricle. The aneurysmal sac bulges well into the right ventricle. The article is well illustrated, and contains numerous references to articles upon this subject.

Reynolds: Female, seventeen years old. No previous illness. Died suddenly after exertion. Aneurysm of one of the sinuses of Valsalva

about the size of a hazelnut. It had ruptured by a recent slit-like opening into the right auricle.

Larkin: Male, thirty-five years old. Large aneurysm of the sinus of Valsalva around the opening of the posterior coronary artery. No rupture.

As would be expected, *a priori*, the number of aneurysms of the sinus of Valsalva greatly predominates in the male. As great confusion exists in the nomenclature of the aortic sinuses, it is impossible to show in tabular form from which sinus the aneurysm originated. It is best to consider that there are two posterior leaflets and but one anterior.

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November 12, 1896.

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28. *Anomalies of cardiac valves.*

Exhibited by JOSEPH SAILER, M.D.

The first specimen was removed from W. B., white man, aged forty years. The clinical diagnosis has been lost, and I only remember that no mention was made of any cardiac condition. The pathologic diagnosis was: Cyanotic induration of the kidneys, with acute hemorrhagic nephritis. Hypostatic congestion and emphysema of the lungs. Chronic gastritis. Chronic endocarditis and fatty degeneration of the myocardium. The following are the notes made upon the heart: The visceral pericardium was infiltrated with soft, yellow fat, and the subpericardial vessels were slightly tortuous. The right auricle was of normal size; its endocardial lining was pale, and some marantic clots were found in

the auricular appendix. The tricuspid orifice admitted three fingers. The leaflets were slightly thickened at their edges, and the right leaflet was adherent to the ventricular wall for about 7 mm. from the auriculoventricular septem, the adherent portion being yellowish. The endocardium of the right ventricle was soft and about 6 mm. thick. It was largely composed of fat, and there was some fibrous change in the muscle. The left auricle was of normal size; the mitral orifice admitted two fingers; the mitral leaflets were thickened, and toward their bases were a few patches of atheroma. The chordæ tendineæ were long and slender; the papillary muscles thickened at the tips, and there were two fibrous bands crossing the ventricle. The cavity of the ventricle was of about normal size, the wall 15 mm. thick, and the structure of the myocardium looser than normal. The aortic orifice was closed by two leaflets; the posterior measured 35 mm. in length; it was soft, with a thickened, fibrous ridge about ½ mm. from the edge; the sinus of Valsalva was greatly dilated. The anterior leaflet was 32 mm. in length; the upper edge was thickened, smooth, without corpus Arantii; the ridge of contact is about 3 mm. from the edge, and the tissue of the leaflet was slightly thicker than that of the posterior leaflet. The sinus of Valsalva was divided at about the middle by a low, fibrous ridge that passed from a small fold in the aorta to the middle of the leaflet; on either side of this ridge were the orifices of the two coronary arteries, both of which were smooth and round. At the right junction of the valves they were adherent for about 7 mm. from the wall of the artery, the line of union being thick and hard, and continued into the cavity by a smooth, hard nodule, or papilla. On the left side the leaflets were not adherent. The walls of the coronaries were soft; the intima was smooth. Two small masses of fibrin were adherent to the aorta some distance above the valves; otherwise the intima was normal. Viti (*Lo Sperimentale*, 1886) has described two specimens in which this condition was present; in one there was a distinct raphé between the two coronaries as in the present case, and in the other the two leaflets were only partially united. Lawrence has, however, reported a case in which the coronary arteries arose from different segments; in this case, although the leaflets were very unequal in size, there was no raphé or other sign of fusion. André Berger has also reported a case with apparent fusion of two aortic leaflets. Peacock, in 1851, was able to collect thirty-two cases with this deformity, but found that it was usually associated with some other serious malformation, leading to early death.

Regarding the etiology of this condition, two theories have been advanced—faulty development and inflammatory change. The latter

is at least indicated by the frequent occurrence of the thickening in the center of one of the segments. But the hypertrophy of the other valves indicates the very early occurrence of the condition; and it must not be forgotten that left-sided fetal endocarditis is of even greater rarity than a double aortic valve. If, however, we admit the coalescence of two of the three embryonic cushions, it is easy to realize that a slight ridge might indicate the line of junction.

Anomalies of excess are more common. The two hearts presented come from two patients, one of sixty-one and the other of sixty-six years, so that the condition is by no means associated with any tendency to curtail life. Lawrence, Rolleston, and Viti have all reported specimens, all characterized, as are these, by the fact that one of the valves was rudimentary. This is the common anomaly of the pulmonary valves, but Wynne Foot has reported a case of two pulmonary leaflets without raphé or other sign of union. Of course, in these cases the embryologic explanation is the only one acceptable.

November 12, 1896.

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29. *Aneurysm of abdominal aorta; rupture into left pleural cavity.*

Exhibited by Dr. Frederick A. Packard.

The patient from whom this specimen was removed was a large, muscular negro, aged forty-six years, admitted on September 10, 1896, on account of retention of urine. He gave a history of gonorrhea at the age of twenty-four years, but no other history of his past appears in the notes. Owing to the presence of an impermeable urethral stricture situated about three inches behind the meatus, a perineal section was performed for immediate relief, and the stricture was divided.

Recovery from the operation was progressing favorably and the patient was walking about the ward when, on October 31st, he suddenly fell to the floor, and was dead when the nurse came over to him. The following notes were made at the autopsy: Body of a thin, emaciated colored man. Almost no subcutaneous fat present; organs of abdomen in normal position; appendix vermiformis thickened and doubled on itself; diaphragm on right side at level of fourth intercostal space, on left side at upper border of sixth rib; pericardium contains normal amount of fluid. Left pleural cavity is filled with clear serum and a huge clot, which latter could be removed *en masse*, and formed a

complete cast of the posterior half of the left half of the chest. The heart was pushed to the right and rotated on its axis, so that the apex pointed directly forward. The heart itself was normal. Slight atheroma was present at the root of the aorta. On removing the left lung there was found a ragged opening in the diaphragm close to the spinal column, readily admitting three fingers and giving entrance to a subdiaphragmatic cavity filled with blood-clot and limited posteriorly by eroded vertebræ. The lungs were normal, save for a small cavity filled with putty-like material at the extreme right apex. The spleen was adherent posteriorly, but was quite readily removed. Small white areas were seen on the surface, which were found to extend for a short distance into the pulp. Scattered through the latter were a few nodules resembling in appearance miliary tubercles. A few adhesions existed between the upper surface of the liver and the diaphragm. The liver itself was normal. The stomach and intestines were normal. The mesenteric arteries were much thickened. The left kidney was pushed far downward and entirely out of its normal position, and the pancreas was pushed far forward. On slitting up the abdominal aorta and inserting the finger, a large cavity was entered. This cavity straddled the vertebral bodies and extended as far as the finger could reach in all directions. It was almost filled with clotted blood, with which were mingled rough bone *débris*. The renal arteries were free from disease, but seemed to spring from the lower extremity of the aneurysmal sac. The aneurysm had dissected the pillar of the diaphragm, and through this could be followed up through the perforation into the left pleural cavity. The psoas muscle on the left side was pushed forward by the mass, and a portion of the muscle had apparently been destroyed posteriorly. The bodies of the eleventh and twelfth dorsal and the first and second lumbar vertebræ were much eroded, those of the two dorsal vertebræ having been entirely consumed and their intervertebral disk standing out as a well-preserved shelf. Almost complete rotation of the pelvis and lower extremities upon the upper portion of the trunk could be accomplished, the only bony connection remaining between the eleventh and twelfth dorsal vertebræ being the articular and spinous processes. At the bottom of the cavity the spinal dura was plainly seen, apparently unaltered.

The anterior wall of the aneurysm is shown in the specimen. It is difficult to explain the man's ability to maintain the erect posture or to walk with such extensive vertebral destruction, while the absence of pain severe enough to cause complaint is remarkable.

December 10, 1896.

30. *Bicuspid pulmonary valve.*

Exhibited by Dr. Frederick A. Packard.

The patient from whom this specimen was removed was a male, Italian, aged forty-five years. He died of pneumonia ten days after admission to hospital. At autopsy the heart was found to weigh 8½ ounces. The right auricle was distended with currant-jelly clots, and also contained some adherent antemortem clot in the neighborhood of the appendix. The foramen ovale was closed, the endocardium of the right auricle normal. The tricuspid orifice admitted three fingers, and was guarded by normal leaflets. The pulmonary orifice was guarded by two valve-leaflets, one measuring three cm., the other four cm. along the free margin. The larger leaflet is fenestrated and its edge slightly thickened. At about the center of the sulcus, behind this larger leaflet, there is a rudimentary septum, hardly traceable so far as the base of the leaflet. *December* 20, 1896.

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31. *Mitral and tricuspid stenosis.*

Exhibited by D. J. McCarthy, M.D.

Mary D., aged forty-six years, single, housewife.

Family history. Negative.

Personal history. Usual diseases of childhood; was of exemplary habits; denying alcoholism and venereal history. For fourteen years had been troubled with dull, vague pains in the joints, in which for the last twelve years there was considerable deformity. The arms were flexed, and there was partial anchylosis of the wrist. The elbows were also partially anchylosed at an angle of 105°. The fingers showed the characteristic deformity of rheumatoid arthritis, with the enlargement of the end of the bones and the trophic changes so often noted in this disease. The knees were only slightly involved, and the ankles, while showing considerable change, were not nearly so deformed as the upper extremities. The heart and lungs were very interesting. The cardiac dulness was markedly increased, the apex being well to the outside of the nipple-line. The apex-beat was diffuse and was associated with a thrill, which, however, was more distinctly noted above the nipple-line. There was a

distinct double mitral murmur. A diastolic murmur at the aortic cartilage was also noted.

There was an effusion into the right pleura, which was tapped and eight ounces of fluid removed; there was diminished liver-dulness; marked edema of legs and some ascites. The patient developed severe epistaxis a week after admission, finally became cyanosed; much venous pulsation, pulmonary edema, and death. The urine showed slight traces of albumin with epithelial and blood casts.

Postmortem. Pleural effusion; lungs normal; cirrhosis of liver; chronic contracted kidneys, and cardiac dilatation, with mitral and tricuspid stenosis. *January* 17, 1897.

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32. *Aneurysm of the celiac axis.*

Exhibited by D. J. McCarthy, M.D.

J. S., aged forty-seven years, born in Virginia, barber, married.

Family history. Negative.

Personal history. Usual diseases of childhood; gonorrhea at eighteen; no chancre; never had rheumatism; used tobacco very moderately, alcohol very moderately.

Status præsens. Last September, while carrying a heavy chair, felt a sudden pain in the abdomen; thought no more of it and went on working; has felt poorly for last two weeks; for last four days has had persistent vomiting; this is his main trouble.

Examination shows emaciation; lungs normal; heart examination negative. A pulsating tumor in the left hypochondrium slightly to the left of the median line. There is a bruit heard, and dulness over this area on percussion. Patient died suddenly twenty-four hours after admission.

Diagnosis. Ruptured abdominal aneurysm.

Postmortem. Abdominal cavities: free clotted blood in general peritoneal cavity, which evidently leaked from the lesser peritoneal cavity through foramen of Winslow. The lesser peritoneal cavity was filled with clotted blood from the rupture anteriorly of an aneurysm (sacculated) of the celiac axis. The sac of the aneurysm was the size of an orange, and the opening of the celiac axis into the aorta was enlarged to the size of a "quarter." The rupture occurred in the upper and anterior portion of the sac. *January* 17, 1897.

33. *Malignant endocarditis following gonorrhea.*

Exhibited by JOSEPH MCFARLAND, M.D.

Of the clinical history of the case nothing could be determined.

Autopsy notes. Body of a white man of average development, aged about forty-five years. The skin and conjunctiva intensely yellow. Eyes open; pupils dilated and directed forward. No rigor mortis. Duskiness of decomposition at lower part of abdomen.

The body was opened by the usual longitudinal section. The organs were found to occupy their proper respective positions. The abdominal tissues appeared bile-stained; the colon was greatly distended with gas. The vermiform appendix and all the dependent portions of the intestine were blue from mingled decomposition and hypostasis. The diaphragm was at the fourth rib.

Thorax. Costal cartilages show considerable ossification. On both sides there are numerous fresh pleuritic adhesions, and especially on the left side some old fibrous adhesions.

Pericardium. Normal in appearance; a slightly increased amount of fluid.

Heart. Rather larger than normal. On incising the cavities, both ventricles contain clots, that of the right being quite intimately attached to the muscular structure. From the auricles dark fluid blood escapes on incision.

On opening the heart, the right ventricle is found to be occupied by a large thrombus that extends through the pulmonary orifice into the pulmonary artery as high as its bifurcation. The thrombus was, without doubt, antemortem, and consisted of dense, stringy fibrin with softened areas here and there.

The thrombus had a point of attachment to the interventricular septum, which, when separated, revealed a patch of endocarditis as large as a quarter dollar. This area was intensely red, and, from the center a group of yellowish points elevated the endocardium and showed that the deeper tissue was occupied by a purulent infiltration. Less pronounced points of endocarditis were here and there to be observed, but were not conspicuous.

All of the valves of the heart were normal. There seemed to be no endocarditis of the left side of the organ.

Lungs. Both lungs were large and deeply congested. In each a number of nodules, varying in size from a pea to an egg, could be felt.

The lower lobe of the right lung was solidified, as in croupous pneumonia.

Section of the lung showed a few miliary tubercles and some tubercular induration of the upper lobe, and showed the nodules to be areas of deep congestion and induration. The first appearance suggested broncho-pneumonia. The cardiac thrombus, however, suggested septic embolism, and the nodules were carefully inspected. Some were found to be fresh; the solidified lung tissue deep black-red in color; others, somewhat older, inclined to be more anemic; others looking much like the gray hepatization of croupous pneumonia, and still others with suppurated central portions. The more isolated infarctions clearly revealed their nature, but when they were massed together, as in the lower part of the right lung, their nature was decidedly obscure.

In addition to the infarctions, the entire pulmonary tissue was congested and subject to hypostatic congestion and decomposition.

Abdomen. The omentum and mesentery showed nothing abnormal.

Spleen. The spleen was much enlarged, measuring 7 x 4 x $1\frac{3}{4}$ inches. It was adherent to the abdominal wall and organs by fibrinous adhesions. Except for the adhesion, the surface was smooth, dull, and glazed. There seemed to be no infarctions in the organ. On section, its substance was pale, anemic, bacony in appearance.

Kidneys. The condition of both kidneys was similar. The organs were larger than normal; soft (decomposition?); the capsule smooth and readily stripped off. Beneath the capsule numerous gray points like small tubercles and some deeply congested points as large as peas were observed. On section, the renal substance was pale and swollen, evidently from cloudy swelling. The congested areas seen beneath the capsule were found to be fresh infarctions; the gray points, older infarctions in which suppuration had occurred.

The adrenals showed no signs of disease.

Bladder. Normal; contained considerable smoky-looking urine.

Generative organs. The penis was phimotic; the narrow opening of the prepuce being closed by a crust. When this was removed, it was apparent that the man was suffering from an acute urethritis. A large amount of creamy pus escaped from the urethra. There was also double orchitis. The prostate gland was not enlarged.

Rectum showed no signs of disease.

Duodenum and stomach. No signs of disease discovered, though the exact conditions were obscured by the advanced decomposition.

Bile-ducts were not patulous. The contents of the gall bladder were thickened.

Liver. Showed fatty infiltration, and is slightly enlarged in consequence.

Pancreas appeared cloudy, swollen.

The intestines were too decomposed to be proper subjects for examination. The only peculiar thing observed was the black-blue discoloration of certain of the dependent portions, causing them to look gangrenous. As has been said, decomposition was too advanced to allow the condition to be accurately studied.

At the apex of the right thoracic cavity, beneath the pleura, there was an abscess the size of a pigeon-egg.

The case seems to be one of the rare cases of general sepsis from gonorrhea, with death from embolic, septic pneumonia, following septic thrombosis and endocarditis of the right ventricle of the heart.

The first natural inquiry would be whether the septic condition was due to the gonococcus or some other organism. The case was, however, unfortunately, in a condition entirely beyond satisfactory bacteriologic research. The heart was taken to the laboratory, and the pus from the endocarditic area subjected to microscopical examination, with the result that immense numbers of cocci, some minute bacilli, and some large bacilli were found, and that not a single biscuit-shaped coccus could be discovered. As the tissues were thus shown to be beyond satisfactory research, cultural study was given up.

One thing, however, seems to suggest itself from the microscopic study of the pus, viz., that no gonococci were present, and that the probabilities are in favor of the diseased condition depending upon the very numerous cocci. This is in keeping with a view advanced by numerous observers that gonococci and staphylococci are apt to grow together in the inflamed urethra, the staphylococci readily penetrating the tissues denuded by the gonococcus, and being responsible for the septic conditions.

Sections of the tissues containing the metastatic abscesses showed the cause of the abscesses to be a coccus, probably the same that had been found in the heart. The coccus occurs in masses, suggesting staphylococcus, but the groups seem to be made up of a diplococcus distinctly dumb-bell shaped and not at all like the gonococcus in its morphology.

Further study will be made of the kidney lesions.

January 14, 1897.

34. *Obliterated very large sacciform innominate aneurysm cured by the introduction of snarled gold wire and the employment of galvanism.*

Exhibited by Dr. D. D. Stewart.

The specimen presented is that from G. D., aged forty-three years, white, male; died December, 1896. The patient came under the writer's observation three and a half years ago. He then was suffering from a very large aneurysm of the innominate artery. The sac was very thin-walled, and, externally at least, was certainly unprotected by clot. The patient was regarded as at the point of death. Surgical interference had been thought impracticable by the surgeons who had seen him. The writer introduced ten feet of very fine spirally-wound and snarled gold wire into the sac, and passed through the wire a galvanic anode current of 80 milliampères, for one hour and a quarter. Neither the history of the case nor the details of the operation need be repeated here, these having been reported in extenso in a paper which appeared in the *American Journal of the Medical Sciences* of August, 1896. It is proper to state, however, that the case was one most unpropitious for any treatment. The patient was what in street parlance may be styled a "bum" or "rounder," who was an habitual spirit-drinker. He was a syphilitic; had pronounced aortic and mitral disease, with extensive cardiac enlargement, generalized endarteritis, and had also chronic nephritis. The aneurysm formed a large and prominent swelling at the root of the neck. As a result of the operation, complete solidification of the aneurysm occurred. The patient was about quite constantly until recently, when, as the result of thrombosis of the left middle cerebral artery, he succumbed. A necropsy was made four hours after death. The report, briefly, is as follows:

Body fairly well nourished. Aneurysmal swelling noted in the situation described.

Thorax: The solidified tumor, with clavicle and three ribs on the right, were removed and the thoracic cavity exposed. The clavicle, with three ribs, the ascending and descending aorta, heart, and pericardium removed en masse.

Lungs: Slightly adherent at both apices. The bases showed hypostatic congestion.

Abdominal cavity: The spleen was somewhat enlarged. A section showed it to be intensely congested, with the trabeculæ visible. Stomach normal in size and situation. Small and large intestines normal.

But one kidney could be found. This was considerably enlarged. It was thought to have been placed aside with the other specimens for subsequent examination, but, unfortunately, must have been returned to the body, as it afterward could not be found with the specimens. The liver was somewhat enlarged and deeply congested. The gall-bladder contained fluid bile. The common bile-duct was patulous.

Heart: Pericardium nonadherent. The sac contained a normal quantity of fluid. The heart was very much enlarged; 26.5 cm. in circumference. Length of the interior of the left ventricle, 11 cm. Thickness of left ventricular wall, 2.5 cm. Thickness of right ventricular wall, 1.2 cm. Cardiac muscle was of a dark-red color; no areas of opacity apparent. Aortic leaflets thickened. Above their inferior attachment, for a distance of 1 cm., the leaflets were densely sclerosed. The aortic valve was found to be very insufficient. Superior borders of two of the leaflets were densely sclerosed. A few atheromatous patches were present on the intraventricular surfaces of the aortic valve. Mitral valve was very patulous, thickened, and slightly opaque, and markedly insufficient. The left auricle was dilated with thickened walls; right auricle was normal in size; tricuspid and pulmonary valves were normal. Coronary arteries were patulous. The aorta from its origin was the seat of very diffuse atheromatous degeneration, with small areas of calcification. Internal circumference of the aorta below the aortic valves, 10 cm. The aorta was dilated from its cardiac origin, but a separate and distinctly outlined extensive fusiform dilatation existed from 1½ cm. to the left from the origin of the left subclavian artery, at the junction of the transverse with the descending portion of the aorta, downward, to a distance of 10 cm. This fusiform sac, at its greatest internal circumference, was 15 cm. This dilatation, a typical fusiform aneurysm, was sharply defined above and below by concentric elevated rings or constrictions of the whole circumference of the aorta. The entire aorta, including the dilated portion, was uniformly thickened. The inner surface of the fusiform sac showed widespread atheromatous patches.

The sacculated aneurysm sprang directly from the root of the innominate artery. The aneurysm-sac was approximately the size of a fetal head at term; length, 13 cm.; transverse diameter, 9 cm. The sac was completely consolidated with organized coagula, in which lay the coils of gold wire. The consolidated sac had at its base a small cul-de-sac, the remains of the innominate artery. This, from the aorta, admitted the little finger to a distance of 4 cm. through an annular ring, sharply defined, 2 cm. in diameter. The cul-de-sac was noted to contain in its interior a small coagulum, presumably of postmortem formation.

The sac itself was very firm and wholly solidified, and when cut into was found to be completely occupied by organized material, in the interstices of which lay the coils of fine gold wire.

Brain: A large thrombus was evident in the left middle cerebral artery. Softening had occurred in the region of the corpus callosum, corda, and ventricular nuclei and in the internal capsule on the left side.

Discussion.—Dr. Hare suggested that Dr. Stewart show the photographs of the patient taken during life. The important point is the length of time the patient lived, three and a half years, after the wire was passed into the aneurysm. In cases of this kind the sac usually breaks soon and causes death.

Dr. Stewart added that the aneurysm had seemed to be on the point of rupturing, and when the needle was introduced the wall appeared to be entirely unprotected and the blood spurted out as soon as the needle had passed through the skin. A month afterward he could insert needles for two or three inches at different points without drawing blood—apparently a clot completely filled the aneurysm. The man had not died of the aneurysm, but of the condition of his cerebral vessels and general bad state.

Dr. Daland thought that the introduction of the wire seemed to aid in forming and solidifying clots, whereby the lumen of the vessel was occluded. *January* 28, 1897.

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35. *Aneurysm of the arch of the aorta.*

Exhibited by John M. Swan, M.D.

The pericardium contained 250 c.c. of straw-colored serum mixed with flakes of lymph. The right auricle was full of fluid blood, and the auricular appendage was distended by a large clot. The left auricle contained some fluid blood and a small amount of clotted blood. The mitral orifice admitted four fingers, and the tricuspid orifice admitted five fingers. The pulmonary and tricuspid valves were normal. The aortic valves were thickened, and the mitral valve showed the same change. The arch of the aorta was very atheromatous; the patches were, as a rule, oval in shape, and the majority of them were calcified. The aorta was much larger in calibre than normal. At the junction of the ascending and transverse portions of the arch of the aorta there was

a saccular aneurysm which was filled with a laminated clot. This aneurysm was situated in front of the point of origin of the innominate artery, but, owing to the dilated and lengthened condition of the aorta and the displacement caused by the bulging of the aneurysm, that vessel was given off to the left of the median line. The aneurysm had pressed against the first piece of the sternum, and had caused bone resorption over an area fully 3 cm. in diameter. At one point in this area the erosion of the sternum was complete, and the aneurysmal sac was separated from the skin only by the fasciæ lying over the first piece of the sternum. This point, at the bottom of which the pulsation was to be felt, was to the left of the median line.

The myocardium was much hypertrophied. The wall of the left ventricle was 27 mm. thick.

The heart and the aneurysm weighed 1080 grams.

March 9, 1897.

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36. (*a.*) *Ruptured abdominal aneurysm.* (*b.*) *Ruptured thoracic aneurysm.*

Exhibited by M. H. Fussell, M.D.

Case I.—John W., aged thirty-six years, entered the wards of the University Hospital, August 20, 1896, with the following history:

He was in perfect health up to three months before entrance to hospital; at that time he was struck in the abdomen by the pole of his wagon. Immediately after the accident he fainted and was taken home. He rapidly recovered, and three days after the accident was again at his work of teamster. He worked one month, when he began to have severe abdominal pain, referred to the epigastrium. This pain rapidly increased in severity until he was forced to abandon work.

On admission the face wore an anxious expression. The lungs were normal. The heart's action was rapid but forcible. The apex-beat of the heart was displaced downward and to the left. The epigastrium was the seat of a mass as large as a cocoanut; dull to percussion, painful, fixed, and pulsating. This pulsation was expansile and not transmitted. A double bruit could be heard over the mass.

On the fourth day after admission he became suddenly pale, faint, and almost pulseless, and died in four or five hours of asthenia.

Postmortem twelve hours after death.

Body of a well-nourished male, no scars on the exterior of the body.

There was a considerable quantity of serum in the abdominal cavity.

The *right pleural* cavity was filled with liquid blood and blood clots. The heart was pushed to the left, and the apex was under the fifth rib. No liquid in the left pleural cavity. Both sides of the heart were empty; the left ventricle 2½ cm. in thickness; valves normal. From the middle of the transverse colon to the cecum, occupying the entire right renal region, is a large blood-clot, posterior to the peritoneum and closely bound down by the perinephric cellular tissue. The upper border of the liver was on a level with the fourth rib. The left vault of the diaphragm was pushed up by a fluctuating mass, composed of the stomach, duodenum, colon, and pancreas matted closely together by old adhesions. Surrounding the esophagus and in front of the aorta was a large aneurysm pushing its way upward through the esophageal opening in the diaphragm to near the root of the right lung, where it emptied into the right pleural cavity. The heart and aorta together with the abdominal viscera were removed for dissection.

The heart was greatly hypertrophied. The left ventricle measured three-quarters of an inch in thickness; the right one-quarter of an inch. The walls of the auricles were greatly thickened. The entire thoracic aorta was normal so far as size was concerned; there were a few atheromatous plates in its walls. The innominate was only one-half inch long; both carotid and subclavian came off almost together. The left subclavian came off on the posterior aspect of the arch. In the abdomen there was a huge saccular dilatation of the aorta, beginning seven inches above the bifurcation and ending two inches above the bifurcation, and involving all of the abdominal vessels above the bifurcation except the inferior mesenteric. This sac was filled with a firm laminated clot, the whole right half being practically occluded.

The right renal artery was practically occluded. The right kidney was atrophied. The mass measured five inches from above downward. The walls of the sac were very thick, expect the right side opposite the superior mesenteric artery, and were so involved in the surrounding inflammation that it was with great difficulty dissected. The extreme anterior sac, a little to the left of the median line, contained a cavity free from clot. The posterior part of the sac had eroded the vertebræ, and the sac was torn in separating it from the vertebræ. A rupture had occurred on the right superior part posterior to the peritoneum, emptying into the right perinephric space and forming there a clot of blood, entirely posterior to the peritoneum, which rested over the kidney like a huge cap, eight inches high and four inches broad.

The measurements of the aneurysmal sac were five inches at its greatest width and five and a half inches at its greatest height.

The aneurysm had ruptured posteriorly, emptied into the perinephric space, was confined there, and pushed its way, as above described, to the root of the right lung, where it emptied into the pleural cavity.

Case II. *Ruptured thoracic aneurysm.*—Wm. M., aged fifty years, of good family history. No history of syphilis; drinks considerable beer.

December 23, 1896, he presented himself at the Dispensary of the University of Pennsylvania for treatment, complaining of pain in the right elbow and right shoulder. The pain was worse on movement, worse in damp weather. Only after careful questioning was it discovered that there were slight dyspnea and palpitation of the heart; that simple exertion caused the elbow-pain to increase, whether the joint itself was moved or not.

On examination there was found to be a spot of dulness reaching from the clavicle to the middle of the third rib on the right side, and from the right border of the sternum about two inches to the right. There was pulsation over the dull area and a double murmur; the diastolic murmur was conducted down the right border of the sternum.

The apex-beat of the heart was outside of the nipple-line in the fifth interspace.

The patient was put at rest and given potassium iodid, with great benefit so far as pain and distress were concerned. He disappeared from treatment and went to work. He reappeared in September, 1896, much emaciated, with all his signs much increased.

He very rapidly emaciated, the mass in the thorax increased, and at a date in December, 1896, the note was made that the mass was much more prominent and the second rib was sharply angulated over its center. The apex-beat of the heart was in the fifth interspace outside the nipple-line. He died very suddenly on January 12, 1897.

Postmortem seventeen hours after death. Body much emaciated, almost no fat left in the tissues. The abdominal cavity is filled with purulent serum. Some purulent flakes adherent to the abdominal walls and the intestines. The vermiform appendix is free from adhesions and filled with fecal concretions. The right pleural cavity is filled with liquid, the superior layer being clear serum, the next layer bloody serum, and the last or bottom layer currant-jelly blood-clots; about two pints of such clots being removed. The right lung is pushed upward and forward. The left lung is so tightly adherent to the wall of the chest that it is necessary to use a knife in separating it. The pleura is about one-quarter of an inch thick, and the entire lung is solid with tuberculous infiltrations and contains numerous cavities. In removing

the ribs the aneurysmal mass is found adherent to the ribs. The second rib is sharply angulated and almost eroded through by the pressure from below.

The heart and aorta were removed for dissection.

The heart is scarcely, if at all, hypertrophied. It is soft and, to the naked eye, has the appearance of fatty degeneration. The wall of the right ventricle measured only one-eighth of an inch thick; left, three-eighths to one-half of an inch. The aortic valves were competent. The pulmonary artery is normal; the ductus arteriosus is present and obliterated. The aortic orifice measures only one and one quarter inch in diameter. The coronary arteries are normal.

The aorta measures half an inch above its exit two inches in diameter; two inches above its exit it measures three inches in diameter; three inches above its exit it measures six inches in diameter. Opposite the origin of the innominate it measures two and one-half inches; opposite the left carotid two inches.

Between the left subclavian and the ductus it measures one and one-half inches, and abruptly ends in the normal-sized vessel in the descending portion of the arch. On its anterior aspect the artery is uniformly dilated. On its posterior aspect there are numerous sacculations. The greatest sacculation is from the superior part of the arch, beginning three inches from the heart and ending one inch from the innominate, measuring at its base four inches, and in height three inches from the line of general dilatation of the arch. The wall of the sacculation is of paper-like consistence, and is well filled with laminated clot; impinges on the second rib, which it has eroded almost through; in the upper posterior portion of the sacculation is a rent one and one-half inches long, through which the man bled to death. The internal surface of the artery is filled with numerous atheromatous plates. These measurements are all made from the collapsed artery. There is another dilatation on the posterior surface just below the origin of the three vessels, which is the size of a hen's egg.

In Case II. the thoracic aneurysm was interesting from several points of view.

It will be noticed that the only symptom of which the patient complained was pain in the right arm. The pain was worse on motion, worse in damp weather, and might easily have been considered rheumatic in origin, as it had been indeed by a number of physicians, if a physical examination had not been made. It was only after the lesion was discovered and pointed questions asked that the patient admitted dyspnea and palpitation of the heart.

The absence of hypertrophy of the heart was also rather remarkable when it is considered that the aneurysm had lasted so long a time. The patient was under observation one year, and, of course, the lesion had lasted much longer.

Both radials were necessarily the same, as the dilatation embraced both subclavians about equally. There was no paralysis of the vocal cords because the recurrent laryngeal did not pass over the part of the artery affected.

The case would have been an excellent one for an attempt at cure by the introduction of wire, as the sacculation was so marked, and in it the rupture took place.

The conduction of the diastolic murmur is of interest. It was conducted down the *right* side of the sternum exclusively, and could not be heard on the left side. It is true that in this case the diagnosis was perfectly clear, but if the mass had not been so evident the case might have been mistaken for aortic incompetency. There was no aortic regurgitation, as proved by the fact that the hydrostatic test showed conclusively that the aortic valves held without any leak.

It has seemed to me, as taught by this and other cases, that the conduction of a diastolic murmur down the *right* side of the sternum would be one valuable diagnostic sign in separating aneurysm of the arch from aortic regurgitation.

Case I. was of but three months' duration, yet the hypertrophy of the heart was considerable. So great was the hypertrophy, indeed, that at the first I was inclined to give some weight to the thought that the aneurysm had either existed before the traumatism, or that there was some other cause for the hypertrophy; but when the atrophy of the right kidney and the hypertrophy of the left were viewed in connection with the monstrous disturbance of all the abdominal circulation, I believe that the hypertrophy was due to the aneurysm alone. The situation of the large clot posterior to the peritoneum, and the subsequent course of the blood over the aorta and through the space surrounding the esophagus in the diaphragm and final leakage, was of the greatest interest. Notwithstanding the enormous rent in the sac, the death was comparatively slow, owing to the partial stoppage of bleeding from the pressure by the perinephric clot.

An excellent idea of the enormous dilatation in Case I. can be had by comparing the calibre of the arch in that case with the arch in Case II., which is comparatively normal. *February* 25, 1897.

37. *Patulous ductus arteriosus.*

Exhibited by A. Hand, Jr., M.D.

The patient, aged, I believe, seven months, with congenital cyanosis, from whom this heart was removed was admitted to the baby ward of the Children's Hospital, under Dr. Griffith's care, and died of a bronchopneumonia. This red string passes from the left ventricle out through the aorta; the white one lies in the right ventricle and pulmonary artery, while the probe passes from the aorta through the large ductus arteriosus and pulmonary artery into the right ventricle.

March 11, 1897.

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38. *Ulcerative endocarditis.*

Exhibited by Joseph McFarland, M.D.

I am, unfortunately, unable to give the Society any clinical history of the case before us. The specimens reached me from an unknown source.

They consist in a hypertrophied and somewhat dilated heart, the chief interest about which centers about the aortic valves, which are thickened, calcified, and nodular. The posterior valve has a fenestrum of considerable size, the edges of which are infiltrated and ulcerated. The aortic valves are only two in number, the divisions between the leaflets having been extinguished, probably, by the process of the disease. Indeed, so altered are the valves that it is difficult to make out just where one stops and another begins.

The aorta, beyond the sinuses of Valsalva, is dilated almost to an extent required to be called aneurysmal, and has, in the centre of the relaxed portion, what seems to be a defect in the wall. Whether this is an accidental injury done with the postmortem instruments or whether it pre-existed is difficult to say. It looks like an arteritis.

The spleen of the same case is greatly enlarged, and contains several infarcts.

The kidney is cirrhotic, and shows numerous small retention cysts.

The phleboliths come from the pelvic veins of a case of malignant disease of the pelvic organs. They represent calcified venous thrombi. They are about the size and shape of grains of wheat.

Discussion.—Dr. W. E. Hughes said that the sequence was stricture, pus-kidney, as the result of passing sounds, and, finally, malignant endocarditis. The most interesting fact was that the patient had moderate pulmonary tuberculosis and an old chronic endocarditis, which had existed together for a considerable time. The malignant endocarditis had been engrafted on the old process. *March* 25, 1897.

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39. *A note upon the condition of the blood-corpuscles in tri-nitro-benzol poisoning.*

Exhibited by Alonzo Englehart Taylor, M.D.

I beg to lay before the Society for inspection the stained blood drawn from the capillary circulation of a man suffering from tri-nitro-benzol poisoning. Of the clinical history nothing concerns us here, except the fact that the identity of the poison ingested was determined by chemical study of the contents of the flask from which the man drank the poison. During the period of coma the color of the skin was a deep purple, and at that time the blood exhibited the absorption-bands of methemoglobin very strongly.

It was at that same time that these coverglass-preparations were made. The noteworthy point about them is that the neutrophilic granulations of the polymorphous leukocytes are purely oxyphilic, refusing entirely the neutral mixtures, and staining with all acid aniline stains as clearly as normal eosinophilic granules. Twenty-four hours later, when the brunt of the intoxication had passed away, this curious anomaly had disappeared ; the blood then stained normally. I cannot explain the phenomenon, but must explain that tri-nitro-benzol applied to coverglass preparations of normal blood will not produce it; it must be a chemicophysiologic alteration. A noteworthy point is that the red corpuscles in this blood were, in all respects, normal in number, appearance, and staining reactions; this fact suggests that the toxic action of the poison upon the red cells occurs in some organ or organs, and not in the general circulation.

March 25, 1897.

40. *A case of tuberculous pericarditis.*

Exhibited by THOMPSON S. WESTCOTT, M.D., and D. L. EDSALL, M.D.

The patient from whom this interesting specimen was removed was first seen December 1, 1896. She was a colored woman, aged about fifty-four years, but much older in appearance, who had always enjoyed good health and had been able to follow the occupation of a laundress until the preceding summer, when she had what was said to be an attack of heat-stroke, of not very severe grade, but from which her recovery had been slow, and she had gradually been forced to give up active work. For several weeks before I saw her she had suffered from what she thought was a "cold," and had seen a neighboring physician several times, who had prescribed for her at his office and visited her at home. Nothing in her personal history or in her surroundings gave any indication of the pathologic process afterward discovered in a study of the diseased organs. When I first saw her she had general anasarca; there was slight fever; cough, with occasional muco-purulent expectoration, dyspnea, and prostration. The temperature was about 101.6° in the evening; pulse, 110, and respirations rather labored, numbering 32 to the minute. Physical examination showed marked hypertrophy of the heart, but the sounds were weak, and no murmurs were detected; the radial arteries, which were alone palpable at the time on account of the edema, were markedly atheromatous. Apart from a few mucous râles, the lungs showed no signs of disease, except at the base of the left lung, where the usual signs of a moderate fluid effusion, reaching, posteriorly, well up to the interscapular region, were apparent. The urine was diminished in quantity and contained considerable albumin and a few hyaline and granular casts. No pain in the chest had ever been complained of. During the first week under treatment the general edema subsided, the temperature gradually fell, the urine became more copious, and the pleural effusion markedly diminished; but, following this temporary improvement, there was a recrudescence of fever, increase in the effusion, and consequent dyspnea, together with diminution in urine excreted, and gradual, but progressive, signs of uremic intoxication. On account of the age of the patient and the evidence of failure of the renal functions, a grave prognosis was given, and an exploratory puncture of the chest was made to determine the nature of the effusion, which had now persisted, but with decided vari-

ations in the physical signs indicating its amount from day to day for nearly three weeks. Clear serum, distinctly blood-stained, was obtained. As her facilities at home for operative treatment were limited, I advised removal to hospital, and she was accordingly admitted to the ward of my colleague, Dr. John B. Roberts, at the Methodist Hospital, where she passed from under my personal observation. The subsequent history can be briefly told. On the day of her admission, December 22d, 8½ ounces of blood-stained serum were removed by aspiration from the left pleural cavity. On the 28th, 28 ounces were removed, and again, 18 ounces three days later. From this time she gradually failed, refusing food and becoming noisy and restless, and, though temporarily improving under treatment addressed to her renal disease, died in uremic coma nineteen days after her admission.

The autopsy was made by Dr. Edsall, acting pathologist of the hospital, and revealed, beside the conditions recognized clinically during life, a somewhat rare and pathologically interesting condition of the heart, which has warranted its exhibition this evening. Dr. Edsall has made a careful study of the pericardium, and has been able to establish the fact that the old pericarditis and presumably the pleurisy was of tuberculous origin.

The body was that of an aged and emaciated colored woman.

The peritoneal cavity was found normal. The stomach and intestines exhibited no lesions. The liver was a little large; the fibrous tissue increased; the parenchyma pale. The spleen was slightly large; firm; somewhat congested. The kidneys were firm; the capsule stripped with difficulty, tearing bits of tissue with it; the cortex was contracted, rather fatty in appearance. Pancreas and suprarenals normal.

Chest. In the right pleural cavity, a few parietal adhesions; strong and complete adhesions with the pericardium. No exudate. The right lung was much congested posteriorly at the base. No other lesions.

In the left pleural cavity, about 100 c.c. bloody fluid. Strong adhesions at the apex to the sides of the chest, the diaphragm, and especially to the pericardium. The pleural surface covered with a thick, fibrinous exudate. The left lung also congested at the base; and at the apex, slight induration.

The parietal pericardium adherent to lungs and diaphragm; thick and inelastic, and everywhere adherent to the visceral layer, except over a circular surface of two inches diameter on its anterior surface. The free space thus formed contained about 20 c.c. bloody fluid. The two surfaces of the pericardium were here covered with a thick layer of

blood-stained fibrin, and elsewhere firmly bound together with adhesions so strong as to require dissection with knife and scissors. On section through the heart-walls they were found covered with fibrin, ¼ to ½ inch thick; the walls much hypertrophied; the cavities all dilated; the heart-muscle rather pale, but quite firm. The valves showed no abnormalities, excepting slight thickening and retraction of the mitral leaflets.

Microscopic examination of both layers of the pericardium showed a round-cell infiltration, with typical tubercles lying all along the surfaces beneath the fibrin layer. Clumps of round-cells, with the centers filled with detritus, and within and without the tubercles numbers of giant-cells of large size and containing a great many nuclei. A few tubercle bacilli were found. The heart-muscle was rather poorly stained; the striations very indistinct, and the muscle-fibres had a blurred appearance. There was a good deal of nuclear multiplication, and a moderate number of leukocytes were seen between the muscle-fibers. *April* 8, 1898.

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41. *Buttonhole mitral and tricuspid regurgitation. Multiple emboli of the lung.*

Exhibited by D. L. Edsall, M.D.

The patient was a carpenter, aged sixty-two years, who was sent in with a diagnosis of nervous asthma, to Dr. James Hendrie Lloyd's wards at the Methodist Episcopal Hospital. He had all the signs of the lesions found after death, except the emboli. Cardiac dulness much enlarged laterally; presystolic and systolic thrill; long drawn murmur of rough quality, presystolic and systolic in time, and a loud first sound. Second pulmonary tone accentuated and reduplicated; positive pulsation of the cervical veins, and a strongly pulsating liver. Anasarca and cyanosis. He became stupid and delirious in a few days, and died two weeks after admission. He spat no blood and had no lung signs beyond very numerous râles.

At the autopsy we found in the abdomen a slight effusion of clear liquid; the stomach and intestines normal beyond small hemorrhages in the mucosa; the liver large, congested, tissue pale; the spleen moderately enlarged, fairly firm from increase of fibroid tissue, much congested; the kidney capsules stripped readily, the tissue somewhat pale, and the line between cortex and pyramids indistinct.

In the chest: Slight effusion in both pleuræ; posterior portion of right lung much congested, one infarct the size of a walnut in the middle lobe; the lower part of the upper lobe and the whole lower lobe of the left lung hard, and occupied almost entirely by seven infarcts, from the size of a cherry to that of an orange.

The pericardium contained a moderate amount of clear fluid. There was no pericarditis. The heart was much dilated in all cavities, with but little thickening of the walls. The cavities were filled with chicken-fat and currant-jelly clots. The tricuspid orifice was much dilated; the posterior valve was thickened and contracted at the edge. The mitral orifice was 5 mm. by 2 c.m. in size and surrounded by a stiff ring of calcareous tissue, which was very rough on the auricular surface, and had some small clots clinging to it. The contracted remains of the mitral valves, the chordæ tendineæ and muscular papillæ attached thereto were bound down to the walls of the left ventricle. The pulmonary and aortic valves were normal. The brain and spinal cord could not be examined. *April* 8, 1897.

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42. *Segmentation and fragmentation of the myocardium.*

Presented by Ludwig Hektoen, M.D.,
PROFESSOR OF MORBID ANATOMY IN RUSH MEDICAL COLLEGE, CHICAGO.

Introduction. The muscular fibers of the mammalian heart were shown by Eberth[1] to be columns of short striated and nucleated cells or segments held together at the ends by a delicate cement. The cells have lateral processes that coalesce, by means of cement, with those of adjacent cells. Normally the layers of cement between the ends of the cells and their processes are not visible; on the addition of acetic acid the cement-substance becomes quite plain, and with 30 per cent. solution of caustic potash the individual muscle-cells may be isolated (segments of Weissmann).

Of the morbid changes of the myocardium proper none occur more frequently or under a greater variety of circumstances than the lesion known as segmentation[2] and fragmentation of the muscle-fibers.

[1] Die Elemente der Quergestreiften Muskeln, Virchow's Archiv, 1866, Bd. xxviii. p. 100.

[2] In biology segmentation means cell-cleavage; generally it means the act of dividing into the segments or parts into which any body naturally separates. It is in the latter sense that the word is used in connection with the lesions of the myocardium under discussion, and it means the separation of the muscle-fibers of the heart into the cells or segments of which they

While Virchow,[1] v. Zenker,[2] Rindfleisch,[3] and Coats[4] make incidental mention of transverse separation or fracture of the cardiac muscle-fibers, Renaut,[5] in association with Landouzy, in 1877, was the first to call direct attention to the dissociation of the muscle-cells. They showed that, as they believed, under the influence of general nutritive disturbances the intercellular cement softened and the individual cells were set free. They referred to this condition as "disintegration, dissociation, segmentation—*état segmentaire.*" This observation was followed by a number of clinical and anatomic studies of the subject by pupils of Renaut as well as by other French writers — Colrat,[6] Durand,[7] Chalot,[8] Weill,[9] Dejerine,[10] Duplaix,[11] Mollard.[12] Mention of the change was also soon made by various French systematic writers—Pitres,[13] Lancereaux,[14] Jaccard,[15] Paul,[16] Robin,[17]—and in 1890 Renaut[18] published an article in which he not only describes the anatomic changes, but also gives the clinical picture of the condition, which he dignifies as an independent disease. The principal symptoms are: Arhythmia, weakening of the impulse of the apex, some increase in the area of the heart's dulness, an uncertain systolic murmur, and, occasionally, edema of the lower extremities. He attributes the dissociation of the cells to solution of the cement-substance on account of senile involutionary changes, alcoholism infection, and the associated high temperature. Individuals affected with such changes in the heart-muscle may perish on account of syn-

are composed. "Fragmentation" signifies a breaking into irregular pieces. Both terms are applied to dissociation of the cardiac muscle fibers. In this paper they will be employed as consistently as possible.

[1] Ueber parenchymatöse Entzündung, Virch. Arch., Bd. iv. p. 266.

[2] Ueber die Veränderungen der willkürlichen Muskeln in Typhus Abdominalis, 1864.

[3] Lehrbuch der pathologischen Gewebelehre, 1870, p. 202; 4th ed., p. 239.

[4] Two Cases of Calcareous Infiltration of the Muscular Fibers of the Heart, Glasgow Medical Journal, 1872.

[5] Note sur les alterations du myocarde accompagnant l'inertie cardiaque, Société de Biologie, July 7, 1877.

[6] Contribution a l'étude des myocardites chroniques et la désintegration cardiaque, Lyon Méd., 1879.

[7] Etude anatomique sur la segment cellulaire cardiaque, Thèse de Lyon, 1879.

[8] Essai sur la désintegration de la fibre musculaire, Thèse de Paris, 1880.

[9] De la mort subite dans la pleurésie, Revue de Médecine, 1887, No. 1.

[10] Sur les altérations du myocarde come cause de mort subite dans le fièvre typhoide, Soc. de Biol., Dec. 26, 1885.

[11] Contribution a l'étude de la sclérose, Arch. Gén. de Médecine, 1885, 7 série, p. 145.

[12] De la myocardite segmentaire essentielle et principalement de la forme senile de cette affection, Thèse de Lyon, 1889.

[13] Des hypertrophies et des dilatations cardiaques indépendantes des lésions valvulaires, 1878.

[14] Traité d'anat. path., 1881, p. 799.

[15] Traité de path. interne, 1883

[16] Maladies du coeur, 1883, p. 468.

[17] Leçons de clinique et thérapeutique médicale, 1877, 20ième leçon.

[18] Gazette des Hôpitaux, 1890, p. 202.

cope or asystole, or from intercurrent diseases which, otherwise of little importance, assume a much more serious aspect on account of the existing heart-lesion.

In 1888 Przewoski and Klein[1] described some cases of very sudden death which were attributed to extensive myocardial segmentation. The following year Browicz[2] published a study of segmentation based on the careful examination of forty-two cases. This is the first publication on the subject in the German language. He concludes that segmentation is due to changes in the cement-substance, occurs ante mortem, and may cause cardiac insufficiency and arrest of the heart's action.

In the discussion upon the disturbances of the myocardium at the Tenth International Medical Congress in Berlin, in 1890, v. Recklinghausen[3] states that microscopic appearances and the frequent connection of dissociation with sudden or rapid, spontaneous death lead him to regard this change in the cardiac fibers as the result of excessive stimulation and irregular contraction of their substance, which passes directly into rigor mortis, and is accompanied with a separation into segments as well as a breaking into fragments. There is, therefore, no ground for believing, as Renaut does, that the primary change is in the cement substance. The frequent association of these changes with the various pathologic conditions included under the term myocarditis shows that these conditions, on account of changes in the muscular tissue or in the heart's activity, predispose the fibers to dissociation.

v. Zenker[4] agrees as to the agonal character of actual dissociation. It would be impossible for cardiac contractions to continue after a general disintegration in this manner of the muscle-fibers; but it is equally evident that a heart whose fibers undergo agonal dissociation must have been the seat of intravital, physical, and chemical changes that gave rise to the weakness and final arrest. The *état segmentaire* is, therefore, an anatomic change that explains to a certain extent many deaths from "heart-failure" (*Herzschlag*).

Marchand[5] (Marburg) in the same discussion questions the statement that the fragments always correspond to the heart muscle-cells, and suggests that possibly the lines of separation represent, as assumed by Wagner, irregular lines of contraction along which separation later on occurs.

Tedeschi[6] found that in two hundred and thirty-six deaths from various

[1] Gazeta lekarska, 1888, No. 18.

[2] Ueber das Verhalten der Muskelzellbalken des Herzens in path. Zuständen, Wiener kl. Wochenschrift, 1890, No. 80.

[3] Verhandlungen des X. Intern. med. Congress, Berlin, 1890, Bd. ii. Abth. 3.

[4] Loc. cit.

[5] Loc. cit.

[6] Ueber die Fragmentatio des Myocardium, Virch. Arch., 1892, Bd. cxxviii. p. 185.

causes segmentation was present in 48 per cent. The frequency of dissociation in infectious diseases and in acute and chronic diseases of the central nervous system led this author to believe that physical changes in the cement, due to toxic substances or to nutritive disturbances, are the essential causes of the process.

Israel[1] made the important observation that in districts of separation there is a marked loosening in the secondary bundles, due apparently to stretching of the intermuscular connective tissue—a change that would point to the action of mechanic influences. Not being able to produce any changes by means of mechanic overdilatation or stretching of dead hearts, he assumes the constant presence of some deviation from the normal in the primitive bundles of disunited hearts, such as pigment atrophy, parenchymatous changes, and fatty degeneration. Under such conditions he is inclined to conclude that any increase in the work placed upon the heart may lead to separation.

Oestreich,[2] in 1894, reviewed dissociation or fragmentation with reference to the anatomic changes, to its relation to the cement, and to its importance as regards the heart's work. He came to the conclusion that it concerns not so much separation of the muscle-fibers into muscle-cells by a solution of the cement, as spoken of by the previous authors, but that fragmentation is essentially, as this term would imply, multiple fractures or tears of muscle-fibers, the lines of which oftener pass through the muscle-cells than along the cement-substance. Longitudinal ruptures were also occasionally seen. Such being the case, and fragmentation occurring very often in otherwise normal hearts in persons dying suddenly, rapidly, or slowly from known causes, he concludes that it is caused by irregular contractions during the death-agony, and may occur in connection with practically all diseases and modes of death.

L. Bard[3] regards the *état segmentaire* of Renaut as fragmentation or rupture of the fibers rather than a segmentary dissociation. These ruptures are probably of postmortem origin, due to reagents and diverse manipulations; yet they are not artefacts, because the real condition is an excessive fragility on account of secondary nutritive troubles. It is, therefore, a subordinate lesion, found in grave acute diseases and in asystolic hearts, that cannot be promoted to the rank of a special primary process.

[1] Zur Entstehung der Fragmentatio Myocardii, Virch. Arch., 1893, Bd. cxxxiii. p. 551.

[2] Die Fragmentatio Myocardii, Virch. Arch., 1894, Bd. cxxxv. p. 79.

[3] De la signification anatomique et clinique des diverses lésions du myocard, Congrès Français de Médecine (1894), Paris, 1895, p. 806.

Renaut,[1] at the first French Congress of Medicine, in 1894, again reviews the subject of segmentary dissociation of the myocardium, and essays to prove, in contradiction to v. Recklinghausen,[2] Oestreich,[3] and others, that it concerns an undoubtedly vital and not merely agonal lesion; that segmentation and fragmentation are part and parcel of a complex and progressive, definite, morbid process, the seat of which is in the muscle-cells. This process is characterized anatomically by great swelling or gigantism of the nuclei of the muscle-cells, by a marked increase of the intercontractile plasma of the cells, which causes an atrophy of the fibrillæ—"hyperplasmic atrophy"—making the fibers perhaps brittle, and thus predisposing to fragmentation; and, finally, by softening and liquefaction of the intercellular cement, which allows segmentation to take place. This dystrophy, of whatever cause it may be, leads sooner or later to cardiac asthenia, and Renaut claims that senile cardiac insufficiency due to this pathologic process is susceptible of clinical diagnosis.

American medical literature is silent upon the subject of myocardial dissociation. The scattered remarks on the subject in the *Twentieth Century Practice* contain historically erroneous statements that deprive Renaut of his due credit as the one to whom we owe our knowledge of the lesion.

The essential basis for the present consideration of this form or forms of disturbance in the myocardium rests partly upon a study of the literature bearing on the subject, but essentially upon the results obtained from investigation of a large number of hearts from animals and human beings dying from a variety of causes and under diverse circumstances.

Artificial Segmentation. The authors are now practically agreed that segmentary dissociation is not of postmortem origin, neither is it an artefact. v. Recklinghausen,[4] Renaut,[5] and others have found well-marked segmentation in hearts from bodies—especially of executed individuals—that were examined immediately after death. I have found it present in bodies examined within one hour after death.

Histologists are well aware, especially since Eberth's[6] work, that certain reagents, such as solutions of acetic acid, have to be applied to render the cement-substance visible; and, furthermore, that 30 or 40 per cent. solutions of KOH dissolve the cement and set the muscle-cells free.

Tedeschi[7] found that the fixing and staining fluids ordinarily in use

[1] Sur la dissociation segmentaire du myocarde, Congrès Français, etc., 1895.
[2] Loc. cit. [3] Loc. cit. [4] Loc. cit.
[5] Loc. cit. [6] Loc. cit. [7] Loc. cit.

do not produce misleading artefacts. Flemming's solution only seemed to render the cement-substance visible, and should consequently not be used in studying the heart-muscle in regard to the presence of segmentation.

While segmentation was looked upon as a cadaveric change by O. Israel,[1] in 1889, he freely denies all influence of artificial and post-mortem factors in its production in 1893.[2]

Rindfleisch claims to have produced mechanic disintegration by over-distention of a dead rabbit's heart, but neither Israel[3] nor Browicz[4] could produce segmentation by mechanic overdilatation and stretching of dead hearts. Israel[5] found that forcible overdilatation of tetanized hearts did not produce dissociation.

While decomposition under ordinary circumstances (Browicz,[6] Tedeschi[7]) does not cause dissociation, Dunin[8] found that artificial gastric juice as well as decomposition under water may cause separation of the cells. He, therefore, regards chemical changes in the organism as the essential cause of dissociation, while mechanic conditions might produce ruptures of the muscle-cells themselves. He expresses the opinion that possibly the agonal invasion of the bacterium coli commune may cause chemical solution of the cement substance.

A number of human hearts were allowed to decompose in a moist atmosphere, at room-temperature as well as in the ice-box; but it so happened that none of them, examined at irregular intervals up to ten days after removal from the body, showed any separation of the fibers. Pieces from healthy human hearts were also allowed to decompose under water as well as to remain for from twenty-four to forty-eight hours in bouillon-cultures of colon bacilli; but the fibers did not become disunited, not even in the vicinity of the bacterial colonies in the interior of the pieces. Similar results were obtained with animal hearts. In this respect my observations differ from those reported by Dunin.

Animal as well as human hearts were examined before and after rigor mortis, but no difference in the structure of the muscle-fiber was observed. Hearts were allowed to freeze stiff, but segmentation did not occur. The view expressed by Rosenbach,[9] that the fall of the temperature of the body after death leads to dissociation, did not receive support.

[1] Praktikum der path. Histologie, 1889, p. 255. [2] Loc. cit. [3] Loc. cit.
[4] Ueber d. Bedeutung d. Veränderungen der Kittsubstanz der Muskelzellbalken des Herzens, Virch. Arch., Bd. cxxxiv. p. 1.
[5] Loc. cit. [6] Loc. cit. [7] Loc. cit.
[8] Einige Bemerkungen über die Fragmentation der Herzmuskelzellen, Ziegler's Beiträge, 1894, Bd. xvi.
[9] Die Krankheiten des Herzens und ihre Behandlung, p. 666.

It may, therefore, be concluded that true segmentation, as ordinarily observed, is not an artefact or postmortem change. In reality there is all reason to believe that no artificial separation of the cells can, under any circumstances, present such microscopic pictures as the sections of hardened specimens of hearts the seat of typical dissociation (Fig. 5). On the other hand, it cannot be denied that various reagents and manipulations may produce ruptures of the muscle-fibers, especially if the latter are brittle on account of degenerative changes. Thus, pressure on the cover-glass may be sufficient to produce breaks in underlying fibers in teased and unimbedded specimens.

Experimental Segmentation and Segmentation in Animals. Tedeschi[1] instituted an extensive series of experiments; but infections, injuries to the brain, traumatic sudden death, overstimulation, etc., failed to produce dissociation in mice, guinea-pigs, rabbits, cats, and dogs. Cutting of the vagus produced indications of segmentation in circumscribed districts. Some segmentation also occurred around wounds made with red-hot needles.

Oestreich,[2] having observed segmentation in persons that died from or in chloroform-narcosis, tried in vain to produce this change by killing animals (four dogs and one rabbit) by administration of chloroform.

Charrin[3] showed that among the extensive changes in the myocardium following the injection of the sterile products of the bacillus pyocyaneus are necrotic areas, in the center of which there may be segmentation of the fibers.

J. Mollard and Cl. Regnaud[4] found that in dogs that die from diphtheria-intoxication sinuous lines corresponding to the points of contact of the muscle-cells at times become visible, and that occasional segmentation may take place. Fractures of the fibers were also present.

I have examined a large number of hearts from dogs, cats, chickens, rabbits, guinea-pigs, mice, and rats that died from various infections or were killed in the course of ordinary laboratory-experiments (here is included a number of animals that died from experimental air-embolism). The results were practically negative as regards segmentation. Only in one guinea-pig that died from diphtheria intoxication did the heart present distinctly visible transverse cement-lines.

The fresh hearts were obtained from six hogs, six sheep, and six young cattle butchered at the stock-yards. Segmentation was not present in

[1] Loc. cit. [2] Loc. cit.

[3] Verh. d. X. Intern. Med. Congresses, 1890, Bd. ii. Abth. 2.

[4] Lésions du myocarde dans l'intoxication aigue par la toxine diphthérique, Annales d. l'institut Pasteur, 1897, t. ii. p. 97.

a single heart; not even were the cement-lines visible. All the hearts were firm and healthy.

A thorough histologic study was also made of the different portions of ten horse-hearts. The hearts were obtained from old and useless horses that were killed for the products obtainable from their bodies. In almost all these hearts the cement-substance was quite distinct, in the form of transverse, quite straight, more or less prominent lines; but typical segmentation was not observed in a single instance. The pigment was very plain in some of these specimens.

Through the courtesy of Dr. A. H. Baker, of the Chicago Veterinary College, I obtained the heart of a horse that died of enteritis and the heart of one dying from influenza, pneumonia, and exudative pleurisy. Both these hearts were the seat of well-marked parenchymatous degeneration, the consistence being rather soft and the cut surface presenting a yellowish-gray, opaque appearance, and the sections showed some separation of the muscular fibers in the right as well as in the left ventricle, but this separation seemed to be due more to a rupture of the fibers than to solution of the cement, because the margins of the fragments were very ragged.

It is consequently probable that in the hearts of diseased larger animals segmentation and fragmentation also occur, although perhaps not to such an extent as these changes are observed in man. It would seem that, on the whole, human heart-muscle is especially prone to dissociation as compared with the heart-muscle of animals.

Segmentation and Fragmentation in Man. The hearts from more than two hundred persons dying from all sorts of causes have been studied as regards segmentation and allied changes. Many of these hearts were obtained through the courtesy of Dr. Louis J. Mitchell, physician to the Coroner's office of Cook County, and I wish to thank Dr. Mitchell for his great kindness in this matter, especially because he made it possible for me to examine the hearts from those dying suddenly from natural as well as traumatic causes. I also wish to thank Prof. J. B. Herrick and Dr. Noel for aid in obtaining suitable hearts. The large majority of the specimens came from the dead-house of the Cook County Hospital.

As far as possible accurate observations were made in regard to the time of examination after death, to the degree of contraction and of fulness of the different cavities of the heart, and especially as to the looseness or firmness of texture of the heart's flesh, in order to obtain, if possible, any data that would warrant the formation of an opinion as to the presence of segmentation from the mere naked-eye inspection.

In the majority of the cases teased preparations from the left papillary muscles and from the ventricular septum were made in physiologic salt-solution and mounted in glycerin. Taking the statements of Tedeschi and Oestreich for granted, namely, that if fragmentation is present at all in a heart, it will be found first in the left papillary muscles and then in the septum, it was concluded that if the teased specimens failed to demonstrate segmentation, then no further examination of such hearts would be made except to harden, embed, and make stained sections from the two places mentioned, in order to control the correctness of the conclusion based on the examination of the teased preparations. It was found that practically, without any exception, the study of carefully and gently made teased specimens will demonstrate satisfactorily the presence or absence of general segmentation. Small pieces in which the fibers run parallel with each other should be used and carefully dissociated by means of fine, clean needles. Teased specimens are hardly sufficient, however, to demonstrate the absence or presence of focal segmentation, because the routine histologic examination of a number of specimens in which teasing had not revealed any changes showed frequent foci of segmentation. Whenever the teased specimens showed fragmentation, suitable pieces were cut out from the different parts of the hearts and hardened in 5 per cent. solution of formalin in alcohol, or in Müller's fluid, embedded in celloidin or paraffin, and stained (after fixation on the slide in the case of paraffin specimens), mostly with hematoxylin and eosin. A few fatty degenerated hearts were fixed in Flemming's solution and stained with safranin. I can confirm Tedeschi's statement that none of the ordinary histologic methods give rise to misleading artefacts. Like Tedeschi, I also observed that the intercellular cement becomes markedly prominent in preparations fixed in Flemming's solution; but inasmuch as it concerned mostly fatty hearts, usually with some real dissociation, I cannot say how much of the apparent change was real and how much artificial.

The following statement contains in condensed form the results of this study:

TABLE I.—LIST OF 125 CASES OF MYOCARDIAL DISSOCIATION.

Age	Sex	Diagnosis.	Bacteriologic examination.	The heart.	Microscopic examination of the myocardium.
1½	F.	Bronchopneumonia, acute nephritis.	0	Flesh pale.	Cement visible, some separation.
14	M.	Crushing injury; quick death.	0	Firm.	Cement visible, some segmentation.
19	M.	Gunshot-wound of heart; quick death.	0	Flabby.	Slight fragmentation.
19	M.	Gas-asphyxia.	0	Quite firm.	Focal segmentation and fragmentation.
20	M.	Bullet-wound of spleen; slow death.	0	Flabby.	Cement visible, focal segmentation.
21	M.	Bullet-wound of abdomen; peritonitis.	0	Firm.	Intercellular cement distinct.
21	M.	Tetanus.	0	Firm.	Segmentation, fragmentation, degeneration.
21	F.	Peritonitis after laparotomy for pelvic abscess.	0	210, firm.	General segmentation with ruptures, degeneration.
22	M.	Constitutional syphilis, transverse myelitis, cystouretero-pyelonephritis.	0	220, brown, firm.	Segmentation, blood between the segments.
23	M.	Died while scuffling.	0	Dilated, loose in texture.	Marked segmentation, with diastasis.
23	F.	Lobar pneumonia, acute and chronic metritis, endocarditis, chronic nephritis.	Pneumococci in the blood.	480, firm.	Occasional dissociation fibers in left papillary muscle.
23	M.	Gunshot-wound of the head.	0	Firm.	Segmentation and fragmentation.
24	M.	Chronic pulmonary and interstitial tuberculosis.	0	Atrophic, firm.	Cement visible, pigmentation.
25	F.	Epileptic idiot, status epilepticus.	0	Firm.	Moderate segmentation.
25	M.	Pneumonia, arteriosclerosis, cloudy swelling.	Pneumococci in blood.	380, quite soft.	Degeneration, some rupture.
25	M.	Typhoid fever, mitral stenosis.	Typhoid bacilli in all organs.	290, firm.	Segmentation, fragmentation, and degeneration.
25	F.	Puerperal peritonitis.	0	Flabby.	Segmentation and fragmentation.
26	M.	Lobar pneumonia, acute nephritis.	0	410, firm.	Slight segmentation.
26	M.	Acute ulcerative endocarditis.	0	400, quite firm.	Typical segmentation, large nuclei.
26	M.	Drowning.	0	Quite firm.	General segmentation.
27	M.	Chronic aortic endocarditis, myomalacia cordis, acute pericarditis, chronic nephritis.	Staphylococci in all organs.	640, mottled.	Focal segmentation, chronic fibrous changes.
27	F.	Strychnin-poisoning.	0	Flabby.	General segmentation.
27	F.	Puerperal eclampsia.	Colon bacilli in all organs.	220, firm.	Some segmentation.
28	M.	Lobar pneumonia, acute nephritis.	0	Firm.	Focal segmentation in left papillary muscle, perivascular thickening.
28	M.	Lymphosarcomatosis.	Sterile.	Small, empty, firm.	Focal segmentation, pigmentation.
28	M.	Herniotomy, rupture of aortic aneurysm into left auricle; sudden death.	Sterile	450, firm.	Segmentation, fragmentation, arteriosclerosis, giantism of nuclei.
30	M.	Drowning.	0	Firm.	Occasional dissociated fiber.
30	M.	Pyemia.	Streptococci and staphylococci everywhere.	Abscesses in myocardium.	Segmentation with disarrangement, leukocytes between segments.
30	F.	Puerperal eclampsia.	Sterile.	Flabby.	General segmentation; cloudy swelling, large nuclei.
30	M.	Lobar pneumonia, acute pericarditis.	Pneumococci in lungs and kidneys.	410, yellow spots.	Segmentation in left papillary muscle, large nuclei.

Age	Sex	Diagnosis.	Bacteriologic examination.	The heart.	Microscopic examination of the myocardium.
30	M.	Acute miliary tuberculosis, tuberculous leptomeningitis	0	Normal.	General segmentation.
30	F.	Chronic nephritis, chronic mitral endocarditis, cirrhosis of liver.	0	.....	Segmentation, large nuclei.
30	M.	Gunshot wound of femoral artery.	0	Firm.	Focal segmentation.
30	F.	Sarcoma of pons.	Sterile.	Firm.	Cement distinct.
30	M.	Appendicitis, peritonitis, pneumonia (coma), chronic nephritis.	0	Firm.	Segmentation in left papillary muscle.
31	M.	Atrophic pancreas, diabetes.	0	250, firm.	Cement-lines distinct.
32	M.	Typhoid fever, peritonitis.	Colon bacilli in the blood.	320, flabby.	Focal segmentation in left papillary muscle.
33	M.	Erysipelas, septicemia, defect in ventricular septum.	Staphylococci in all organs.	480, firm.	General segmentation, large nuclei.
33	M.	Chronic parenchymatous nephritis, anasarca.	0	600, firm.	Segmentation, arteriosclerosis.
33	M.	Sarcoma of left cerebral hemisphere.	Sterile.	440, firm.	Segmentation, some sclerosis.
35	F.	Peritonitis after laparotomy.	Streptococci and staphylococci in blood.	280, firm.	Segmentation.
35	M.	Carbolic-acid poisoning.	0	Flabby.	General segmentation.
35	M.	Chronic interstitial and pulmonary tuberculosis, tubercular meningitis.	0	360, soft.	General segmentation, coronary sclerosis.
35	F.	Epileptic convulsion.	0	270, firm.	Segmentation in left papillary muscle, some sclerosis.
35	M.	Cerebral hemorrhage.	0	Firm.	Cement-substance visible.
35	M.	Pernicious malaria.	Malarial organisms in the blood	370, flabby.	Moderate focal segmentation.
35	F.	Fracture of skull, alcoholism	0	Quite firm.	Fragmentation, some vascular changes.
36	F.	Pulmonary and interstitial tuberculosis.	0	125 brown, firm.	Cement distinct.
36	M.	Endomyopericarditis, arterial sclerosis.	0	600, firm.	Some segmentation.
36	M.	Lobar pneumonia.	Pneumococci in all organs.	420, firm.	Severe segmentation, degeneration.
37	M.	Typhoid fever.	Staphylococci and colon bac. in all organs.	380, flabby.	Segmentation, arteriosclerosis.
37	M.	Cerebral hemorrhage.	0	Loose in texture.	General segmentation, large nuclei.
38	M.	Traumatism of brain; instantaneous death.	0	Normal.	Segmentation in left papillary muscles.
38	F.	Mitral stenosis.	0	Firm.	Segmentation with diastasis, fibrous thickening.
40	M.	Pulmonary and laryngeal tuberculosis.	0	Muscle soft, pale.	General segmentation with diastasis, some fibrous thickening.
40	M.	Urethral stricture, perineal abscess, septicemia.	0	Flabby, loose.	Segmentation in left papillary muscle, some fibrous thickening.
40	M.	Rupture of urethra, cystouretero-pyelonephritis.	Colon bac. in kidneys.	250, yellowish.	Cement occasionally visible.
40	M.	Died while drunk.	0	Firm.	Moderate dissociation, some sclerosis, large nuclei.
40	F.	Chronic interstitial nephritis, chronic fibromyocarditis, chronic aortic and mitral endocarditis.	0	450, firm.	Occasional dissociation, chr. int. myoc., large nuclei.

Age	Sex	Diagnosis.	Bacteriologic examination.	The heart.	Microscopic examination of the myocardium.
40	F.	Peritonitis after removal of uterine appendages.	Colon bac. in blood.	250, flabby.	Moderate segmentation, blood between segments, cement distinct.
40	F.	Acute and chronic endocarditis.	0	Large heart.	Dissociation, degeneration, fibrous changes, large nuclei.
40	F.	Chronic nephritis, meningeal hemorrhage.	0	290, soft	Some dissociation, fibrous changes, large nuclei.
40	M.	General arteriosclerosis.	0	450, firm.	Segmentation
41	M.	Cirrhosis of liver, chronic nephritis.	0	Large, quite firm.	Some segmentation, increase in fibrous tissue, fatty degeneration.
42	F.	Arteriosclerosis, fibrous myocarditis, chronic aortic endocarditis.	0	Firm.	Cement visible, some segmentation.
43	M.	Carcinoma of stomach.	0	Firm.	Cement plain.
43	M.	Arteriosclerotic aneurism, rupture.	0	Dilated, soft.	Extensive segmentation, bacilli in myocardium.
44	F.	Constitutional syphilis, chronic nephritis.	Sterile.	540, firm.	Dissociation, fibrous increase, large nuclei.
45	F.	Carbolic-acid poisoning.	0	Firm.	Segmentation with disarrangement of segments.
45	M.	Carbolic-acid poisoning.	0	Firm.	Cement plain.
45	F.	Arteriosclerosis, chronic endocarditis, uremia.	0	320, firm.	Segmentation, marked thickening.
45	M.	Arteriosclerosis, chronic nephritis.	0	700, firm.	Extensive segmentation, hyaline vessels.
45	M.	Cirrhosis of liver, nephritis, arteriosclerosis.	0	640, firm.	Segmentation in left papillary muscles, advanced sclerosis, large nuclei.
45	M.	Lobar pneumonia.	Diplococci in slips. from blood.	380, flabby.	Marked segmentation, degeneration.
45	M.	Railroad crush; chronic interstitial nephritis.	0	Firm.	Focal segmentation in left papillary muscles, scars, large nuclei.
45	M.	Bullet-wound of head.	0	Firm.	Dissociation.
45	F.	Chronic fibromyocarditis, aneurism of septum.	0	750, firm.	Fragmentation and segmentation in left papillary muscles, fibr. myoc., large nuclei.
46	M.	Lobar pneumonia, septicemia.	Staphylococci in blood.	430, flabby.	Advanced segmentation, sclerosis, large nuclei.
46	F.	Chronic endocarditis, chronic nephritis.	0	370, soft.	Advanced segmentation.
46	M.	Lobar pneumonia.	0	300, soft.	Cement broad, segmentation, large nuclei.
46	M.	Arteriosclerosis, chronic nephritis.	0	Large, firm.	Some dissociation, scars.
47	M.	Lobar pneumonia, chronic nephritis.	0	600, firm.	Segmentation, large nuclei.
47	M.	Lobar pneumonia, acute nephritis.	Pneumococci everywhere.	600, mod. con.	Cement broad, occasional segmentation, large nuclei.
49	M.	Chronic nephritis.	0	Flaccid.	Marked segmentation.
50	M.	Cerebral hem'rhage, arteriosclerosis, chronic nephritis	0	880, firm.	Segmentation, chronic fibrous changes, large nuclei.
50	M.	Cerebral hemorrhage.	0	380	Segmentation, chronic fibrous changes, large nuclei.
50	M.	Traumatic rupture of liver.	0	Firm.	Dissociation marked.
50	M.	Fibromyocarditis, aneurysm of left ventricle.	0	700, fibrous	Segmentation, large nuclei.
50	M.	Alcoholism.	0	Normal.	Marked dissociation.
50	M.	Arteriosclerosis; sudden death.	0	Large, firm.	Segmentation, fibrous changes, large nuclei.
50	F.	Crushing injury.	0	Flaccid.	Segmentation, bacilli among segments, much sclerosis.
50	M.	General arteriosclerosis; sudden death.	0	Dilated, flaccid.	Cement broad, marked segmentation in septum, chr. fibrous changes.

Age	Sex	Diagnosis.	Bacteriologic examination.	The heart.	Microscopic examination of the myocardium.
50	M.	Pneumonia, nephritis, cirrhosis of liver.	0	600, firm.	Segmentation in left papillary muscle.
50	M.	Arteriosclerosis, chronic endocarditis, acute pericarditis.	Sterile.	620, firm.	Cement broad, some separation, hyaline vessels, large nuclei.
50	M.	Arteriosclerosis, cirrhosis of liver.	0	580, flabby.	Cement broad, some separation, scars.
51	M.	Bullet-wound of abdomen, peritonitis.	0	Small, fatty.	Focal fragmentation, fatty degeneration, endarteritis, pigmentation.
52	M.	Chronic pulmonary tuberculosis, atrophic cirrhosis of liver.	0	Quite firm.	Slight dissociation.
54	M.	Pneumonia, arteriosclerosis	Pneumococci in blood.	Soft.	Some segmentation.
55	M.	Pneumonia, chronic nephritis.	Pneumococci in blood.	730, flabby.	Segmentation, fibrous changes, large nuclei.
55	M.	Arteriosclerosis, atrophy of kidney.	0	310	Segmentation in left papillary muscle.
55	M.	Aneurysm of aorta, rupture into trachea.	0	530, dilated flaccid.	General segmentation.
56	M.	Carcinoma of stomach.	0	250	Cement-lines broad.
56	M.	Found dead in street, alcoholism.	0	Firm.	Segmentation, endarteritis.
56	M.	Aneurysm of aorta, rupture.	0	Large, firm	Focal segmentation in left papillary muscle, scars, large nuclei.
58	M.	Arteriosclerosis, nephritis.	0	260, loose.	Segmentation, sclerosis of coronary artery.
58	M.	Carcinoma of stomach.	0	Brown, atrophy.	Cement broad.
60	M.	Carcinoma of larynx.	0	300, firm.	Cement-lines broad.
60	M.	Chronic arsenic poisoning.	0	Soft.	Slight segmentation, pigmentation.
60	M.	Pneumonia, meningitis.	Pneumococci everywhere.	450, flabby.	Advanced segmentation, arteriosclerosis, degeneration.
60	M.	Sarcoma of left frontal lobe.	0	550, soft.	General segmentation, cement visible.
60	F.	Pneumonia (peracuta), emphysema.	0	175, brownish.	Late fragmentation, great pigmentation and thickening.
60	M.	Skull fracture, extrav. hem., arterial sclerosis.	0	420, soft.	Segmentation in left papillary muscle.
60	M.	Tubercular peritonitis and pleurisy, chronic nephritis.	0	260, flabby.	Dissociation, large nuclei, vacuolar degeneration.
62	M.	Bronchiectasis, pulmonary gangrene.	0	280, flabby.	Slight segmentation, atrophy, pigmentation.
63	M.	Lipomatosis.	0	Firm.	General segmentation, degeneration.
65	F.	Chronic endocarditis, cirrhosis of liver.	0	380, flabby.	General segmentation, large nuclei, sclerosis, pigmentation.
66	M.	Cerebral hemorrhage, fracture of skull.	0	420, soft.	Segmentation, scars.
67	M.	Emphysema, bronchitis.	0	490, flabby.	Segmentation in left papillary muscle, large nuclei.
68	F.	Pyonephrosis.	0	Firm.	Segmentation in left papillary muscle, large nuclei.
70	M.	Arteriosclerosis, aortic aneurysm, cirrhosis of liver, chronic nephritis.	0	290, soft.	Segmentation, perivasc. thickening.
70	M.	Chronic pulmonary and intestinal tuberculosis, pef. peritonitis.	0	320, brown.	Cement broad, some separation.
70	M.	Contracted kidney.	0	Flabby.	Segmentation, large nuclei.
70	M.	Bronchitis, emphysema.	0	440	Segmentation, large nuclei.
70	M.	Emphysema, bronchopneumonia.	0	Quite firm.	Segmentation, degeneration, chr. fibrous tissue, large nuclei.
70	F.	Aneurysm of aorta, arteriosclerosis.	0	Firm.	Segmentation, hyaline vessels, scars.

A few words of explanation are necessary: The list is arranged according to age in order to bring out prominently the relation of dissociation to the various periods of life. The essential clinical and anatomic diagnosis is stated in order to make plain the connection between dissociation and the various other local and general morbid processes. For the same reason the results of the bacteriologic examination, when made, are stated. The degree of firmness, and the weight of the heart, in grams, are stated for obvious reasons. Finally, in the columns giving the results of the microscopic examination, there is made also a brief statement of other changes present in addition to noting the presence, the degree, and the kind of interruption of the continuity of the muscle-fibers.

In the table showing the percentages I have included under the heading "Segmentation and Fragmentation," in addition to the typical cases, all the instances in which the intercellular substance was plainly visible in the teased specimens, because this change is undoubtedly closely related to actual segmentation, and also all cases of more or less well-marked fragmentation in which it seemed reasonable to believe that the change was not wholly an artefact.

TABLE II.—SHOWING THE RELATIONS OF MYOCARDIAL DISUNION TO SEX AND AGE IN 190 CASES.

Decimal periods.	Normal.		Segmentation and fragmentation.		Percentage of disunion.	
	Female.	Male.	Female.	Male.	Female.	Male.
I.	5	9	1	0	16.66	0
II.	2	4	0	4	0	50
III.	4	9	8	22	66.66	70.96
IV.	3	14	9	19	75	57.57
V.	1	5	7	25	87.50	83.33
VI.	3	1	1	17	25	94.44
VII.	2	3	3	9	60	75
Total . . .	20	45	29	96	59.18	68.08

Total number of cases 190
Disunion in . . . . . 65.78 per ct.

THE FREQUENCY OF SEGMENTATION AND ITS RELATION TO OTHER DISEASES. (*a*) *General Frequency.* v. Recklinghausen[1] found segmenta-

[1] Loc. cit.

tion present in all cases of spontaneous sudden death occurring in the course of chronic myocarditis, coronary sclerosis, or changes at the root of the aorta; in many acute infectious diseases; in cases of sudden death in nephritis with cardiac hypertrophy, but without cerebral hemorrhage; in rapid death from cerebral hemorrhage; and, finally, in many cases of sudden death due to traumatism and other causes. He did not find it in influenza or acute articular rheumatism.

Tedeschi[1] gives the following statistics: In two hundred and thirty-six cases of death from various diseases he observed segmentation or fragmentation in 48 per cent. (in the men 44 per cent., in the women 53 per cent.); twenty-three cases, from one to ten years old, gave 9 per cent.; twenty cases, from eleven to twenty, gave 50 per cent. (43 per cent. in the male and 66 per cent. in the female); fifty-seven cases, from twenty-one to thirty, gave 48 per cent. (43 per cent. in the male and 53 per cent. in the female); forty-eight cases, from thirty-one to forty, gave 54 per cent. (61 per cent. in the male and 45 per cent. in the female); thirty-one cases, from forty-one to fifty, gave 62 per cent. (44 per cent. in the male and 86 per cent. in the female); thirty-one cases, from fifty-one to sixty, gave 40 per cent. (equal in both sexes); fourteen cases, from sixty-one to seventy, gave 80 per cent. for men and 100 per cent. for women; after seventy-six years, 50 per cent. for men and 33 per cent. for both sexes. As regards diseases, severe general infections in adults were accompanied with this lesion in 82 per cent.; deaths after severe operations in 100 per cent. (ten cases); cerebral diseases in adults in 92 per cent.; heart-diseases in 50 per cent. of the cases.

Oestreich,[2] without presenting any figures, says that, after extended observations, it would be practically impossible to name a disease in connection with which he had not observed fragmentation—it may occur in every disease.

In one hundred and ninety consecutive cases that I have examined segmentation and fragmentation in the broadest sense occurred in 65.78 per cent. Except in the first, and, to a less extent, in the second, decennium, this lesion is certainly present in the majority of the cases that die from the ordinary causes under familiar circumstances. In about one-half of the cases it concerned a marked general dissociation.

In the first decennium the only instance in fourteen cases occurred in an eighteen-months-old girl that died from acute nephritis and bronchopneumonia. The cement-substance was clearly visible, and there were a number of ruptured fibers. The other cases examined include a wide range of pathologic conditions.

[1] Loc. cit. [2] Loc. cit.

In the second decennium there were four instances of focal or slight segmentation in ten cases. Two were quick deaths from traumatic causes (crushing injury and bullet-wound of chest), one died from gas-asphyxia, one after a bullet-wound of the spleen, and all were free from any important chronic diseases.

(*b*) *Relation of Segmentation to Infections.* A general bacteriologic examination was made in forty-nine cases. In fifteen the organs and the heart's blood were sterile; in thirty-four the commoner forms of bacteria (the colon bacterium, the pyogenic cocci, the micrococcus lanceolatus, the typhoid bacillus) were found more or less widely distributed. Segmentation and fragmentation were present in twenty of these cases and absent in fourteen. Without entering into details it may be said that any inseparable relation between any single micro-organism and segmentation in general, or at any particular period of life, could not be demonstrated from this small number. Thus, the colon bacillus was present in eight instances, four of which were cases of peritonitis; in three of the eight segmentation was present; the youngest of the five in which it was absent died at fifteen, the oldest at forty-five, both of peritonitis. In ten cases it concerned fatal infection with the micrococcus lanceolatus in the form of an anatomically typical lobar pneumonia frequently associated with endocarditis, pericarditis, and meningitis. In all these cases, the ages of which varied from twenty-three to sixty years, the heart was hypertrophied, weighing from 380 to 730 grams, arteriosclerosis being present in some of the hearts. Segmentation was present in nine. In eight cases of typical fibrinous pneumonia, but without bacteriologic examination, disunion was observed five times.

In eleven cases in which the blood and the organs were sterile, dissociation was observed in five, including sarcomatosis, puerperal eclampsia, constitutional syphilis with interstitial nephritis, and glioma of the brain.

In two cases in particular there were found enormous numbers of rather long bacilli in the generally segmented heart-muscle, scattered between the sides and ends of the segments (Fig. 7). One was killed directly by a wagon passing over the abdomen, the other died instantaneously from the rupture of an aortic aneurysm. This was, therefore, undoubtedly a postmortem bacillary invasion; and the possibility that the bacilli may have dissolved the cement-substance cannot be wholly denied, although it must be remembered that segmentation is observed in cases of sudden traumatic death examined at once. It is evident, however, that the vital, agonal, or postmortem action of bacteria upon

the heart-muscle cannot account for the occurrence of segmentation and fragmentation in general.

(*c*) *The Relation of Segmentation to Diseases of the Heart.* While the number of cases from which deductions concerning the above question can be made is rather insufficient to warrant any extended analysis, yet the frequency with which segmentation occurs in connection with hypertrophy of the heart, due to the usual causes and often independent of the fatal disease, must be emphasized. Thus, in forty-three adult male cases the hearts of which weighed from 320 to 850 grams, segmentation was present in 90 per cent.; in sixteen adult female cases in which the hearts weighed from 270 to 900 grams, segmentation was found in 68.75 per cent.; or in fifty-nine cases of men and women with hypertrophy of the heart, fragmentation was present in 74.57 per cent. More or less extensive chronic fibrous changes, in the broad sense, were present in most of these hearts. In twenty-one adult cases of atrophy of the heart (in women less than 240 grams, in the men less than 290 grams) segmentation, often in slight degree, or merely in the form of visible cement-line, was present in 57 per cent. In all these cases it concerns hearts that were actually weighed.

(*d*) *Relation of Segmentation to Sudden Traumatic Death.* In the list are ten cases of death due to traumatism, such as gunshot-wounds and crushing railroad injuries. In the majority death was practically instantaneous. In all some degree of segmentation was present. Two cases occurred in individuals less than twenty years old.

The Naked-eye Appearances of Hearts the Seat of Dissociation. In "typhoid" and "asthenic" conditions—in which dissociation is so often present—the heart is usually more or less dilated and flaccid, its muscle reddish, brownish, or grayish-yellow, opaque, and friable or loose in its texture. These appearances, however, depend largely upon accompanying parenchymatous degenerations (*myo-degeneratio cordis*). Segmentary dissociation may be present without marked changes in color and consistence. Focal segmentation in particular may be present in the firmly contracted hearts, and von Recklinghausen,[1] Langerhans,[2] and Oestreich[3] describe general segmentation in hearts that were not dilated. Renaut[4] urges the general diastole of dissociated hearts as evidence against segmentation being caused by irregular or violent agonal contractions or ultimate tetanization, as claimed by some, which should leave the heart—in persons examined immediately post-

[1] Loc. cit.
[2] Demonstration eines Falles von Carbolvergiftung, Berl. kl. Wochenschr., 1893, 37.
[3] Loc. cit.
[4] Loc. cit.

mortem—in systole. Segmentation is frequently found in hypertrophic hearts that appear quite firm and contracted. Consequently, changes in color and in consistence, as determined by contraction or dilatation, are not necessarily indicative of dissociation. According to Kaufmann,[1] advanced segmentation may produce wavy swellings on the endocardial surface, especially on the papillary muscles. Oestreich[2] regards increased friability and looseness in texture as very important evidence of dissociation. If, on stroking the cut surface, especially where the fibers run parallel, gently with the knife, clefts and fissures appear numerously in the myocardium, segmentation is quite surely present, even though the heart as a whole may have been quite firm and contracted. This corresponds with my experience. Focal and slight separations, however, require microscopic examination for their demonstration; and in any event it would not be wholly reliable to base a positive diagnosis of general segmentation on the naked-eye appearances only.

The Histology of Segmentation and Fragmentation. Segmentation may present itself in varying degrees. In many hearts distinct, finer or broader, apparently swollen lines, often staining quite deeply with eosin, run transversely across the fibers at irregular intervals (Figs. 1 and 5). The lines may be straight or curved, which is usually the case, or crooked, zigzag, or step-like. Occasionally they extend wave-like through several adjacent and parallel fibers. When diastasis of the fiber has occurred, the substance forming these lines can often be recognized at one of the ends of the separating fiber, while the other end may be smooth or finely irregular or ragged. Incomplete separation along the cement lines is also occasionally observed.

In the twelve instances of this series in which the cement-substance was plainly visible in the form of narrow or broader lines, while segmentation was not presented, it concerned in the majority of cases decidedly atrophic and pigmented or fattily degenerated hearts from adult persons dying from chronic tuberculosis (two), diabetes (one), and carcinoma (stomach, three; larynx, one), the heart in each case weighing less than normal and in one case only 125 grams; but this condition was also observed in persons dying from peritonitis after bullet-wound (one), from sarcoma of the pons (one), from carbolic-acid poisoning (one), from cysto-uretero-pyelo-nephritis following rupture of the urethra (one), and from cerebral hemorrhage (one).

In quite a few cases typical segmentation producing larger or smaller segments with mostly clean-cut ends occurs in smaller or larger foci

[1] Lehrbuch der sp. Path. Anat., 1896, p. 24.

[2] Loc. cit.

and not generally throughout the parts examined. In some of these specimens the cement-lines are visible about the foci of dissociation (Fig. 2). Often a complete cell with its lateral branches is loosened. The width of the space separating the segments may vary considerably. (In some instances foci of segmentation were not found as usual in the

FIG. 1.

Cement-lines visible. Man, aged sixty, carcinoma of stomach. × 125.

left papillary muscles, but elsewhere in the left ventricle; but this was the case only when there were extensive fibrous changes in the papillary muscles.) In some of these cases the heart is otherwise absolutely healthy. In some there are extensive fibrous changes; in some the nuclei are very large, and extensive fatty degeneration may be present. Occasional tears also occur.

From focal dissociation the process may be said to extend through

transition-stages of slight, then more and more pronounced separation, to well-marked, general segmentation throughout the whole left ventricle. In these instances of marked segmentation the change is also usually present in the right ventricle, and in this case it also occurs first

Fig. 2.

Cement broad; some segmentation and, in the centre, a segment partly broken across. Man, fifty-six, arteriosclerosis, rupture of aneurysm into pericardium; hypertrophy of heart. × 500.

and best marked in the papillary muscles. Segmentation is evidently very rarely present in the auricles. The marked frequency with which the papillary muscles are involved is attributed by Israel to absence of antagonistic diagonal fibers.

Typical segmentary dissociation gives rise to segments that correspond quite generally to the outlines of the muscle-cells isolated by means of

solution of potassium hydrate. The ends may be straight, concave, or convex, dentated, or terraced, often finely irregular; lateral processes are present; larger segments, evidently composed of more than one cell, are also found; small square or oblong segments without nuclei, as well as irregular fragments, frequently occur, evidently due to the mechanical fracture of the lateral processes of the loosened segments, but fracture lines very rarely pass near the nucleus (Fig. 3). More or less diastasis of the segments has usually taken place, and the intervening spaces are oftenest apparently empty. Generally the separated pieces maintain a relatively normal position, but sometimes disarrangement has been brought about, due, it would seem, to the contraction of adjacent fibers after segmentation has occurred and to loosening of the interstitial tissues, allowing the bundles to become mixed (Israel[1]). In

FIG. 3.

Marked general segmentation with great diastasis of the pieces. × 120.

occasional cases extravasation of red blood-corpuscles has occurred between the ends of the segments—a fact emphasized by Browicz[2] and others in establishing the vital nature of segmentation. In the vicinity of inflammatory foci leukocytes are found between the ends of the pieces (Fig. 4), and in some cases of ante- or postmortem bacterial invasion the spaces may be occupied by micro-organisms (Fig. 7). Generally the separation is complete. Occasionally, however, there are faint indications

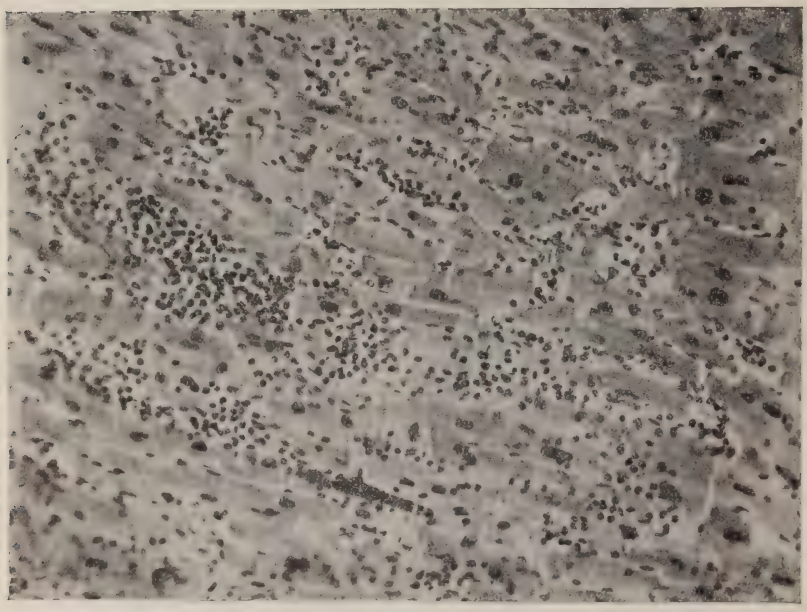
[1] Loc. cit.

[2] Loc. cit.

of the presence of a delicate membrane covering the fiber and still holding the ends together—a sarcolemma, the existence of which in the heart-muscle is, following Eberth,[1] usually denied. Oestreich[2] also notes having observed this delicate membrane. Perhaps the connective-tissue fibrillæ become closely applied to the separating muscle-fibers and give rise to the appearance of a sarcolemma.

Otherwise the section of the heart-muscle may appear perfectly normal, the striations being distinct, the nucleus of usual size, the connec-

FIG. 4.



Acute interstitial myocarditis in pyemia. Near lower margin a compact micrococcal embolus (staphyloc. pyog. aur.). Leukocytic infiltration, segmentation, leukocytes between the pieces. × 500.

tive tissue and bloodvessels unchanged. In many cases the general loosening of the secondary bundles described by Israel is present; or there may be, depending upon general conditions, such as infections and intoxications, parenchymatous and fatty degenerations (Fig. 5) of the sarcoplasma as well as the changes usually included under the term chronic fibrous myocarditis. In a large number of the cases, particularly in

[1] Loc cit.

[2] Loc. cit.

hypertrophy of the heart, with or without chronic myocardial changes, there are noted marked increase in the size and changes in the shape of the nuclei of the muscle-cells. The nuclei may indeed be giants in size. In shape they may be greatly elongated and cylindric or spread out in the form of oval masses; club-shaped, kidney-shaped nuclei, and nuclei

FIG. 5.

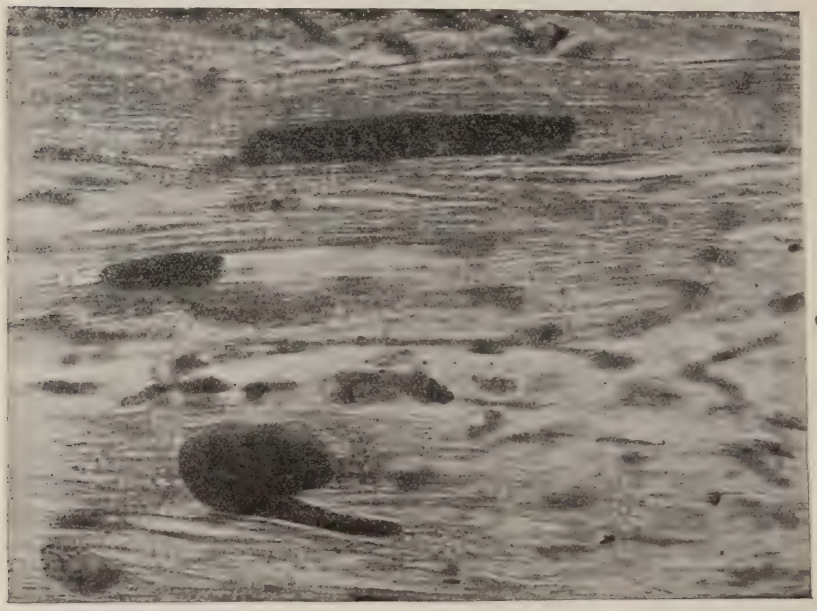
Cement-lines broad and visible in a heart the seat of fatty degeneration × 500.

with budding processes and curiously shaped outgrowths also occur; some are vesicular, others stain very deeply, and sometimes they present surface-markings, due probably to the imprints of pressure, which may give the nucleus an appearance as though it were to split longitudinally (Fig. 6).

Fragmentation, tearing, or breaking of the muscle-fibers, usually present to some extent in all cases of marked segmentary dissociation, as shown by the presence of small fragments, often without nuclei, in some cases occurs to a more marked extent, and may be the predominating form of disunion. The lines of separation may pass through any part of the muscle-cell, often passing around one pole of the nucleus, the margins or ends of fragments being more irregularly ragged than in segmentary dissociation. Incomplete rupture may occur. Fragmenta-

tion, strictly speaking, never occurs to such an extent and does not give rise to separation into such minute pieces as does segmentation. It appears to bear the same general relations to acute and chronic diseases as segmentation, and in agreement with Bard, Renaut, and others the conclusion seems warranted that it is caused largely by mechanic rupture or breaking of the fibers on account of various degenerative changes that render the sarcoplasm brittle.

FIG. 6.



Cylindric and peculiarly shaped giant nuclei; in the center a cell with vacuolar degeneration of its protoplasm. Man, age fifty, heart weighed 700 gr., fibromyocarditis. × 500.

THE DISTINCTION BETWEEN SEGMENTATION AND FRAGMENTATION. In the foregoing the attempt has been made consistently to employ the words segmentation or segmentary dissociation and fragmentation to designate two distinct conditions, each in the main well described by the term applied to it—segmentation referring to the division of the muscle-cells at the lines of junction in the muscle-fiber, fragmentation to the rupture or break of the fiber regardless of the cement-lines.

While Renaut[1] and his pupils, Browicz[2] and others, insist that the

[1] Loc. cit.

[2] Loc. cit.

interruption of the continuity of the cardiac muscle-fibers that gives rise to the characteristic appearances described by them as *myocardite segmentaire* occurs principally along the lines of union between the single muscle-cells, others claim that the lesion is produced essentially by ruptures or breaks of the muscle-fibers without any special reference to the limits of the individual cells.

Browicz regards all interpretations of segmentation that do not make the limits of the pieces correspond with the limits of the cells as erroneous. He does not lay so much stress on the mere dissociation of the muscle-cells, which probably is an agonal product, as upon vital pathologic conditions of the intercellular substance, manifesting itself by the fact that the lines of junction become visible. It may be a homogeneous seam, a broad stripe, or a broader layer whose contours are sharply separated from the ends of the cell. In this space are then found short, stave-like formations running parallel to the long axis of the muscle-fiber (*"breitstreifige, stäbchenformig gebaute Kittsubstanz"*), and such changes may alternate with distinct dissociation. In this statement Browicz has been in a measure substantiated by Przewoski's[1] investigations, which show by special methods that at the ends each muscle-cell has a layer of granules, the *stratum granulosum terminale.* Browicz looks upon these changes in the cement as the initial phase of segmentation.

Eberth[2] long ago called attention to the fact that in atrophic and pigmented hearts the fibers may separate into single cells without any manipulations, and he ascribes this dissociation to fragility of the cement.

Goebel[3] also regards segmentation as beginning with changes in the intercellular cement, and the separation as occurring at the lines of union between the cells.

v. Recklinghausen,[4] Marchand,[5] Oestreich,[6] and Bard[7] lay stress upon the rupture of muscle-fibers as an important step in producing the *état segmentaire* of Renaut. In fact, the last two regard multiple ruptures as the essential occurrence. It concerns fragmentation in the strict sense, and not segmentation, and the process does not depend upon changes in the cement-substance (v. Recklinghausen[8]). Oestreich[9] found that some of the fragments were so small that they could not possibly be whole cells; furthermore, that the lines of rupture very often passed across the fiber in the immediate neighborhood of the

[1] Cited by Browicz, loc. cit.
[2] Loc. cit.
[3] Beiträge zur fettigen Degeneration des Herzens, Centralb. f. allg. Path. u. path. Anat., 1893, Bd. iv. p. 728.
[4] Loc. cit.
[5] Loc. cit.
[6] Loc. cit.
[7] Loc. cit.
[8] Loc. cit.
[9] Loc. cit.

nucleus, and sometimes in a curve around one pole of the nucleus, half of which would then project into the indentation of the corresponding fragment. He also found that the fragments were more like the artificial muscular bits described by Eberth than like isolated muscle-cells. Finally, he was able by means of the action of acetic acid to demonstrate the presence of unchanged cement-lines near rupture-lines. Often the rupture would pass across the fiber near the unchanged lines of union.

While Oestreich[1] is surely correct in the statement that many fragments are too minute to represent whole cells—fragments without nuclei also occur—as well as in the observation that rupture-lines often pass immediately around the nucleus, I have not been able to confirm the statement that the fragments generally resemble artificially produced muscle-morsels nor to demonstrate by addition of acetic acid normal cement-lines in all hearts the seat of marked dissociation. Eberth[2] describes artificial muscular detritus as presenting more clean-cut and more nearly straight ends than the isolated muscle-cells, the ends of which are usually finely irregular, while occasional fibrillæ may project from the ends of forcibly produced fragments, yet the ends of the fibrillæ are not pointed, but cut off squarely. Oestreich uses this statement as a standard in distinguishing between segmentation and fragmentation in human hearts. When making the statement Eberth[3] does not refer to any special species, but to normal heart-muscle in general. Segmentation is a morbid process. There is, therefore, no inherent reason why it may not cause disunion of the muscle-cells and yet the ends be square and clean-cut. For these reasons no weight can be given the conclusions drawn by Oestreich after comparing the segments of dissociation with the artificial fragments described by Eberth.

Histologists—as, for instance, Schweigger-Seidel, in Stricker's *Handbook*—also describe normal muscle-cells with smooth ends, and believe the difference in the limiting surfaces to be occasioned by the circumstance that the muscle sometimes comes under observation in a contracted, coagulated condition. I think it will be found that the ends of fragments of the normal human heart produced by force, pressure, tearing, bending, which I presume is what is meant when Eberth speaks of "artificially produced muscular detritus," are strikingly ragged and irregular, the fibrillæ yielding unequally. Furthermore, in typical, general, marked segmentation, such as is shown in Fig. 3, it is not generally possible to demonstrate any cement-lines. On the other

[1] Loc. cit. [2] Loc. cit. [3] Loc. cit.

hand, the outlines of the segments correspond very nicely to the shape and the size of the isolated muscle-cell, which, forsooth, they often are; nor is it usual to find in such specimens that a line of separation crosses a segment in the region of a nucleus. The study of cases of focal or slight dissociation will also be found to substantiate quite fully the statements of Browicz[1] in regard to the coexistence of visible cement-lines and actual segmentation. On the other hand, fragmentation in the strict sense also occurs, as acknowledged by Renaut,[2] although probably not to the extent claimed by Oestreich,[3] and here the ruptures very often cross the fibers near the nucleus, and hence probably inside the limits of the single muscle-cells. The nucleus, however, is not always central. It may be found quite near one end of the cell. In many specimens it is also possible to demonstrate the coexistence of cement-lines independent of the fracture-lines, the ends on each side of the latter being usually very irregular and ragged, as if the fiber had been torn forcibly across, the fibrillæ offering unequal resistance.

The conclusion is therefore in order that segmentary dissociation as well as actual fragmentation or rupture of the fibers of the heart-muscle both occur.

Distinction should be made between typical segmentary dissociation of the heart muscle-fibers, in which softening or complete solution or perhaps excessive brittleness of the cement allows the muscle-cells and segments to move away from each other, and fragmentation in the strict sense of the word, it which it essentially concerns ruptures of the muscle-fibers in any part of their course and without any special reference to the lines of union. This distinction is quite important, because the possibility that fragmentation may be, to a certain and perhaps large extent, artificial, cannot be altogether excluded, while segmentation cannot be produced by the action of ordinary reagents and manipulations. For these reasons I have preferred to deal more with segmentation than with fragmentation. Segmentation and fragmentation frequently occur in varying degrees. In any large series of hearts it will be found that it is quite feasible to separate the specimens into two groups, according as one or the other of these forms of interruption in the continuity of the muscle-fibers predominates; but true segmentation is by far the more frequent.

The Causes, Nature, and Clinical Significance of Segmentation. The views concerning the nature and the cause of segmentary

[1] Loc. cit. [2] Loc. cit. [3] Loc. cit.

dissociation have, as noticed, failed to harmonize. It was the earlier belief of Renaut[1] that it concerned a primary, nutritive, or chemical change of the cement substance. This view has been supported by his pupils, by Browicz,[2] v. Zenker,[3] Tedeschi,[4] and in part by Israel[5] and Dunin.[6]

Durand[7] and Duplaix[8] claimed that in circulatory disturbances more or less permanent edema of the myocardium may be produced, and the substances contained in the edematous fluid, such as sarcolactic acid, may dissolve the cement, holding the muscle-cells together in the form of muscle-fibers. Durand[9] calls attention to the direct action of very feeble solutions of chromic and other acids upon the cement; but Colrat[10] produced edema of the heart experimentally without the occurrence of segmentation. Segmentation also occurs in many conditions, such as atrophy of the heart, sudden traumatic death, etc., without the presence of edema.

v. Recklinghausen,[11] Oestreich, and Rosenbach[12] look upon the change more as a mechanic disunion and rupture of the muscle-fibers on account of disturbances of innervation and irregular contraction in the death agony, favored perhaps by brittleness of the fibers due to degenerative changes. Oestreich[13] says fragmentation does not cause death, but the last contraction causes fragmentation. Rosenbach[14] states that a maximal contraction precedes dissociation, and that it may be regarded as proved that it is due to the crossing of impulses of different nature in a dying heart. When an impulse of excitation meets an inhibitory impulse, separation of the fiber occurs. Letulle[15] expresses the opinion that segmentary dissociation is due to an agonal imbibition of the muscular fibres with sarcolactic acid. Finally Renaut,[16] in his efforts to overthrow v. Recklinghausen's[17] and Ostreich's[18] views concerning the essentially agonal nature of segmentation, advances the claim that it is the last step of a complex, dystrophic, cellular process, involving the muscle-cells and leading to softening and solution of the intercellular substance.

Lepine and Molard,[19] in 1891, described certain degenerative changes in the heart muscle-fibers as a particular species of nonsclerotic, parenchymatous myocarditis. The changes consist in the appearance of clear

[1] Loc. cit. [2] Loc. cit. [3] Loc. cit. [4] Loc. cit.
[5] Loc. cit. [6] Loc. cit. [7] Loc. cit. [8] Loc. cit.
[9] Loc. cit. [10] Loc. cit. [11] Loc. cit. [12] Loc. cit.
[13] Loc. cit. [14] Loc. cit.
[15] Anatomie Pathologique (Cœur, Vaisseaux, Poumon), Paris, 1897.
[16] Loc. cit. [17] Loc. cit. [18] Loc. cit.
[19] Sur une espèce particulière de myocardite parenchymateuse (sans sclérose), Arch. de Méd. Expérim., 1891, p. 777.

spaces between the primitive fibrillæ, which are thereby forced apart. These spaces are largest near and about the nucleus, which is increased in volume, round, ovoid, or elongated. In consequence of these conditions the fibers, when cut longitudinally, present a bulging enlargement corresponding mainly to the situation of the nucleus. The heart that is described as presenting these changes in typical form weighed 560 grammes, was dilated, and came from a man seventy-eight years old. Segmentation and other lesions are described as absent. Subsequently, however, Renaut, in 1894, calls attention to the fact that segmentation was present in all of Lepine's and Molard's preparations, and, based on further studies, he makes the hyperplasmic atrophy or vacuolar disintegration and gigantism of the nuclei, described by these authors, steps in a complex process leading to segmentation. The enlargement of the nucleus, the increase of the amount of the chromatin, are interpreted by Renaut as evidences of augmented nuclear activity, the muscle-

FIG. 7.

Segmentation; bacilli upon and between the segments; to the left of the center a cell with vacuolar degeneration and containing two nuclei. Man, fifty, died instantly from crushing injury of abdomen. × 500.

segment assuming, as it were, a more intense "nuclear life," while its function and its structure as a contractile element fall into abeyance, and the intercontractile substance is liquefied. *La cellule vit dès lors bien plus cellulairement que musculairement.* Finally the cell softens or

dissolves its cement, which may swell up and become permeable to the corpuscles of the blood and the lymph. The muscle-cell is now functionally incapacitated, and general segmentation is the anatomic sign and final lesion of cardiac asthenia. The "hyperplasmic atrophy" of Renaut, as well as the special form of parenchymatous myocarditis of Lepine and Molard,[1] would seem to correspond very closely to vacuolar degeneration of the muscle-fibers (Figs. 6 and 7), which is due to a colliquative disintegration of the sarcoplasm. It is often associated with enlargement or vesicular tumefaction of the nuclei, and is generally observed in connection with interferences of the coronary circulation (Letulle[2]), and occurs probably also in diverse general intoxications. This condition may or may not be associated with segmentation. Incidentally attention may be called to the fact that spaces in the muscle-fibers of the heart very much similar to these vacuoles have been interpreted as capillaries within the muscle-fibers by Meigs.[3]

Segmentation occurs frequently in association with hypertrophy of the heart, with or without degeneration. Here, also, enlargement of the nuclei, which may assume various shapes, is, as a rule, present. Letulle[4] states that every muscular fiber that hypertrophies presents a proportional enlargement of the nucleus; and Tangl,[5] who made his observations upon the left ventricle of the hearts of rabbits, the hypertrophy being secondary to experimental traumatic lesions of the aortic valve, found the nuclei of the hypertrophic cells of varying shape, mostly oval, sometimes with central constrictions. Often the nuclei were very long, and they seemed in general to enlarge with the cells. Furthermore, the nuclei were surrounded by a granular substance that coalesced with the protoplasm of the periphery of the cell in which small vacuoles were occasionally seen. Evidently the enlargement of the nuclei and their richness in chromatin in purely hypertrophic hearts belong to the hypertrophic process, and cannot in general be assumed to be evidence of any "dystrophy" leading to segmentation. According to Tangl, it is not entirely impossible but that vacuolation of the sarcoplasm may also occur in pure hypertrophy.

Finally, it must be borne in mind that segmentation occurs without "hyperplasmic atrophy" and "gigantism of the nuclei," as witness segmentary dissociation of healthy hearts in connection with sudden,

[1] Loc. cit.

[2] Loc. cit.

[3] The Microscopic Anatomy of the Human Heart, Transactions of the College of Physicians of Philadelphia, 1891; and THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1891.

[4] Loc. cit.

[5] Ueber die Hypertrophie und das physiol. Wachsthum des Herzens, Virch. Arch., 1889, Bd. cxvi. p. 432.

traumatic death, as well as in other conditions. Hence Renaut's claim that segmentation depends upon the existence of a special dystrophic process of the myocardium proper, marked by vacuolar changes in sarcoplasm, by gigantism of the nuclei, and by solution of the cement, is to be regarded as rather broad, first, because segmentation occurs in otherwise healthy heart-muscle; second, because although segmentation occurs generally in hypertrophic hearts, often with secondary degenerative changes, yet it has not been demonstrated that the enlargement of the nuclei observed in such heart-muscle is always dystrophic or degenerative, and not to a large extent progressive and hypertrophic in its nature. The entity of Renaut's dystrophic process is, therefore, still questionable.

Segmentary dissociation occurs so frequently in connection with various acute and chronic myocardial changes as to warrant the conclusion that the cement-substance may be altered, softened, or rendered brittle by direct action of general conditions as well as indirectly on account of primary or secondary lesions of the muscle-cells. Broad, visible cement-layers, focal segmentation, segmentation confined to districts of fatty degeneration in the heart-muscle, dissociation about acute myocarditic foci with leukocytes between the segments, the limited segmentation that occurred around Tedeschi's[1] aseptic wounds of the heart, all point in this direction. Consequently it would seem that v. Recklinghausen's claim that it is not shown that segmentation can occur before the death-agony no longer holds good.

It stands to reason that the changes in the muscle-cells must influence the quality of the cement. This seems actually demonstrated in the following case, instances similar to which I could duplicate:

Ambrosius[2] details the case of a woman, aged forty years, who died ninety hours after the removal of the tubes and ovaries under chloroform. There were fatty degeneration and exquisite segmentation of the heart muscle with vesicular nuclei. The degree of fatty degeneration corresponded nicely to the limits of the different degrees of segmentation, pointing to the presence of a vital, histologic variation in the different fibers, and permitting the inference that dissociation occurs in places prepared for it by changes in the muscular tissue.

As the cement becomes changed focal and general dissociation may occur under normal or abnormal conditions of cardiac activity. Diastasis and disarrangement of the pieces may follow in degrees commen-

[1] Loc. cit.

[2] Ein Fall von spät eingetretenen Tod nach Chloroformiuhalation nebst Bemerkungen zur Fragmentatio Myocardii, Virch. Arch., Supplementheft, 1895, Bd. cxxxviii.

surate with the vigor and the irregularity of the contractions. The coexisting brittleness of the sarcoplasm and presence of loosened segments between still contracting muscular bundles may lead to more or less extensive fragmentation. The conditions present in diseased and especially in hypertrophic hearts favor dissociation. Here are lesions of various kinds of the muscle-cells, of the stroma, and of the blood-vessels that may weaken the cement and the plasma of the cells, and also marked disturbances in the vigor and order of the contractions. It is certainly difficult to account satisfactorily for segmentation in cases of traumatic, sudden death in which the heart-muscle is plainly normal in its structure. In general it may be assumed, however, that dissociation of the muscle-fibers results from a disproportion between the vigor of the muscular contractions and the muscular cohesion. Segmentation and fragmentation may, therefore, occur in normal heart-muscle if its contractions be excessively violent and irregular, as may be the case, for instance, in hanging, in sudden *exitus letalis* during coitus, in excited states, in delirium, etc., and thus become the immediate cause of death because the heart can then no longer contract. In weakened hearts, violent or irregular as well as quite normal contractions may cause dissociation, suspending further cardiac action ; but this cannot be designated a true agonal change in the ordinary sense conveyed by this term.

The views as to the existence of segmentation in a form susceptible of clinical recognition (segmentary myocarditis of Renaut) are divided. Renaut and his scholars claim that in the old the condition occurs in such a typical form that it may be diagnosed, and that it should therefore be regarded as a clinical entity. This view has not gained general acceptance. Primary, independent segmentation is not yet recognized. Aufrecht,[1] in a short article, champions its existence.

Aufrecht describes a case in a man, forty-eight years of age, who died from the general symptoms of heart-failure, with edema of the lower extremities. In the left ventricle there was universal segmentation, with hemorrhage between the segments, which are stated to have been normal with but occasional cloudiness. There was some hypertrophy of the right ventricle, which he interprets as secondary to the segmentation in the left, although the influence of a moderately marked pulmonary emphysema cannot be excluded.

Aufrecht regards this case as one of primary, independent segmenta-

[1] Ueber einen Fall von primärer Fragmentation des linken Ventrikel, Zeitschr. f. kl. Med., 1894, Bd. xxiv. p. 205.

tion of the left ventricle. There exists no ground, he claims, for not believing that a dissociated heart may not work as long as one the seat of parenchymatous degeneration. The muscle-fibers might undergo separation little by little, and the process need not necessarily coincide with the agony.

All the other authors regard general and focal segmentation as an accidental or secondary phenomenon occurring in the course of infections and intoxications in connection with the primary and secondary lesions of asystolic hearts, and with fatal traumatism. It constitutes, as is also shown by the statistics brought forward in this paper, an episode in the course of the principal affection. While it possesses an anatomic individuality, it is so common that it would be difficult to say in what disease it would surely be absent after, say, the twentieth year, and it would take a very long time to enumerate all the diseases in which it has been found present. Its presence may explain this or that detail in the clinical course of a number of processes, such as the termination by sudden death; but it is secondary, it is not a disease in the strict sense any more than cloudy swelling, for instance, and cannot, therefore, be considered as entitled to a place in our nosology.

General segmentation is inconsistent with the continuance of the heart's action. Its presence in cases of arteriosclerosis, coronary disease, fibrous myocarditis, etc., that die suddenly, is consequently a satisfactory anatomical reason for the sudden fatal ending. In medico-legal practice cases of sudden death under more or less suspicious circumstances, or without any apparent cause, occasionally occur in which the most careful postmortem fails to reveal any other explanation of death than general myocardial segmentation, as, for instance, in this example:

An apparently healthy man, twenty-three years of age, was "fooling" with two friends. Two hats were knocked off, and, as one of the men picked up his hat, he observed that his friend—the dead man—had fallen to the floor unnoticed. It was thought that he was shamming, but after waiting three or four minutes it was found that he was dead. At the postmortem, ten hours after death, and made by Dr. Louis J. Mitchell, all the organs, including the brain, were macroscopically normal. The heart, especially the left ventricle, was flabby and dilated. Microscopic examination showed the heart—unfortunately this was the only organ that could be examined—the seat of a general and typical segmentation.

In this case there is not much doubt that the extensive dissociation caused death, but the cause of the dissociation is not clear.

Ramond[1] reports the case of a thirty-year-old professional runner who fell ill after a run of thirty kilometers on a hot July day. There were fever, dyspnea, *delirium cordis*, and anuria, death ensuing in a few hours. The postmortem showed hyperemia of all the organs and a flaccid, dilated heart. Microscopic examination showed absence of myocarditis, but a marked general segmentation and fragmentation of the fibers, with much extravasated blood. The nuclei stained well, and the transverse striæ were distinct. Ramond regards toxins due to muscular overexertion as the cause of the lesions.

While general fragmentation is obviously inconsistent with continued heart's action, focal segmentation need not, it would seem, of necessity, result in death. Browicz concluded that general segmentation leads to sudden death, but focal to cardiac insufficiency, or, possibly, to rupture of the heart or to fibrous substitution of the separated muscle-cells. The last two conclusions he states he is unable to substantiate.

Robin[2] found segmentation along the margins of the ruptures in two hearts in which were also endo- and peri-arterial changes. He looks upon the segmentation as the essential cause. Tedeschi[3] examined the muscle in the vicinity of the ruptures in two hearts. In one instance there were found chronic fibrous interstitial changes as well as scattered foci of fragmentation. In the second case there were no fibrous changes about the point of rupture, but blood-corpuscles were present between the isolated, single muscle-cells. Subsequently Tedeschi had opportunity to examine three additional museum-specimens of spontaneous heart-rupture, with, he states, the same result as in the last case above. From these results it would seem that, while complete details are not at hand, still, in some cases, segmentation and spontaneous heart-rupture stand in such very close relation to each other that the question merits further study.

Of all the parts of the heart, segmentation occurs most frequently in the papillary muscles of the left ventricle. It is often present in a marked degree in these muscles only. If it occur some time before death, the result might be mitral regurgitation, taking it for granted that a dissociated papillary muscle is incapable of contraction, because physiologists in general agree (Landois and Stirling, Curtis, in *The American Text-book*) that it is the function of these muscles, by their contraction during ventricular systole, to hold the mitral valves taut and thus compensate for the narrowing of the cavity of the ventricle. Mitral regurgitation induced in this way may account in some degree

[1] Dissociation segment. du myocard. dans un cas de cœur forcé, Bullet. et Mém. Soc. Méd., 1896; abstracted in Centralbl. f. allg. Path. u. path. Anat., 1897, viii. p. 236.

[2] Loc. cit.

[3] Loc. cit.

for the marked pulmonary congestion so frequently present in cases associated with myocardial segmentation.

If it be assumed that segmentation may occur in foci some time before death, several important questions arise concerning the fate of the loosened cells. Of course the isolated, non-nucleated protoplasmic masses that are often split loose could not maintain their vitality. On the other hand, would the nucleated but dissociated cells necessarily die and become replaced by fibrous tissue? Is the vitality of the muscle-cell dependent upon the integrity of its cement? Could a reunion of the isolated muscle-cells by means of new cement occur? These are questions that cannot be answered at this time. Koester[1] remarks that the yellow degeneration (*Entartung*) which is followed by scar-formation in the myocardium does not, in slight degrees, differ to any extent from the segmentary myocarditis of Renaut. I have studied carefully the appearance of the muscle-fibers in and about myocardial scars, and it has seemed that the fibers, generally speaking, undergo atrophy without segmentation. Segmentation rarely occurs in the muscle-fibers embedded in scars. Any evidences of replacement of muscle-segments by fibrous tissue were not found. It therefore appears reasonable for the present, in view of the uniform absence of any evidence of reactionary changes, to regard even focal segmentation as at the most a product of limited antemortem duration. In conclusion the facts brought forward may be summarized as follows:

The cardiac muscle-fibers frequently separate into muscle-cells and irregular fragments. Segmentation and fragmentation are due to disproportion between the vigor and order of muscular contraction and muscular cohesion. They occur in normal heart-muscle due to excessively vigorous and irregular contractions. More frequently they are encountered in association with acute and chronic, secondary, and primary myocardial changes that alter the cement, weaken the plasma, and predispose to dissociation under normal or increased heart's action. General segmentation is of brief duration, because its occurrence is incompatible with further cardiac contractions. Focal or limited segmentation may, perhaps, cause incompetency of the auriculoventricular valves, especially the mitral. It may lead to cardiac insufficiency, and, possibly, to rupture of the heart. The fate of loosened muscle-cells is not known. It seems, however, that focal dissociation is of but short antemortem duration. The occurrence of primary segmentation as a distinct disease is not proven.

[1] Verh. d. X. intern. med. Congr., Berlin, 1890, Bd. ii. Abth. ii.

43. *A case of endopericarditis.*

Exhibited by S. Solis Cohen, M.D.

M. H., negress, aged fourteen years; born in Virginia. *Family history:* Mother dead, cause unknown. Father alive and well. No history of rheumatism or tuberculosis.

Past history: Says she was never sick a day in her life before.

Present history: Cannot give satisfactory history; but says her knees began to get weak three weeks ago. This was followed by pain and swelling in other joints. A few days ago pain and tenderness began in region of left nipple, and she became short of breath.

Physical examination: On admission to the Philadelphia Hospital, March 27, 1897, a plump, young colored girl, breathing in a labored manner, complaining of pain in precordial region. Some precordial tenderness. A distinct double thrill over body of heart, and on auscultation a loud to-and-fro friction-rub heard best over body of heart and along left border of sternum, whence it diminishes from above downward, being still heard, however, at costal margin to the left of the epigastrium. At apex, which is situated a little out of the midclavicular line in the sixth space, there is heard a soft systolic murmur; this is transmitted a short distance into the axilla, but lost before the midaxillary line is reached.

The knees are swollen and painful; the patellæ floated up; the left knee more affected than the right.

March 28th. This morning the girl has a good deal of distress and dyspnea. Complains of pain in back over right base. The physical signs are only harsh breathing over both bases.

29th. More distress this morning. The endocardial murmur is louder, more booming at apex. Area of cardiac dulness seems larger, though tenderness is so great as to prevent thorough examination; extends from upper border of second rib to costal margin, and from half an inch to the right of the sternum to one and one-half inches beyond the nipple-line. Tenderness in epigastrium and region of spleen. Spleen cannot be felt, but area of dulness is increased. A small area of dulness over left base posteriorly with diminished breath-sounds.

April 1st. Has a great deal of dyspnea; still tosses herself around, and sits up in bed all the time.

2d. Heart about the same. A pleuritic effusion developed in the left pleura, also some in right. Not outlined because of great tenderness.

4th. Called at 8.20 A.M. and found the patient in a great deal of pain; severe dyspnea. She had gotten out of bed and in again alone. Pulse could not be felt. Dyspnea extreme. Skin cold and clammy. Heart tumultuous. Died in twenty minutes from onset of severe symptoms.

Postmortem notes. Autopsy by Dr. David Riesman, pathologist.

Clinical diagnosis: Acute articular rheumatism; pericarditis; endocarditis.

Pathologic diagnosis: Fibrinous pericarditis with effusion; vegetative endocarditis of aortic and mitral valves; hypertrophy and dilatation of heart; localized caseous tuberculosis of right lung and of bronchial and mediastinal glands; cloudy swelling of kidney; fatty infiltration of the liver; bilateral pleural effusion; condensation of lungs; hemorrhagic infarction of both lungs; punctiform hemorrhages of mesentery.

External appearance: The body of a well-developed young black woman with good musculature. Rigor mortis well marked. Several abrasions from blistering over precordium and posteriorly on left side of chest.

Abdominal cavity: The liver extends 6 cm. below the costal margin in the right mammary line. In median line it extends to 2 cm. above the umbilicus. There is some excess of fluid in pelvic cavity, turbid-yellow in color. Mesentery and mesocolon show punctiform hemorrhages. The visceral peritoneum over intestines is free from these hemorrhages. They are also found, in less profusion, in gastrocolic and great omentum. The diaphragm extends to top of seventh rib on left side and to sixth rib on right side.

Thoracic cavity: The left pleural cavity contains 220 c.c. of amber-colored fluid, free from lymph. Right pleural cavity contains 450 c.c. of similar fluid. Left lung is retracted and also right, though less than left. The cardiac surface exposed measures 14 cm. transversely. Right boundary of heart is in right midclavicular line. The apex is in sixth interspace on a line from acromial end of clavicle.

Pericardium: External surface of pericardium is bound by adhesions to right lung. Pericardial cavity contains 300 c.c. of a translucent dark-yellow fluid free from coagula. Pericardial layers are bound together by recent adhesions of lymph. Adhesions vary in length, some are nearly two inches long, and in thickness vary from the size of a thread to one-eighth of an inch in diameter.

Heart: Enormously enlarged and encased in a covering of fibrin, most marked on posterior and lateral surfaces. The auricular appendages are adherent by bands of fibrin. Right auricle contains a large chicken-fat clot, completely filling the cavity. There are adherent clots

in the right ventricle interwoven with the columnæ carneæ, which appear to be increased in number. Thickness of right ventricle is 9 mm. Pulmonary valve is normal. Tricuspid valve is somewhat thickened. The endocardium of right auricle and ventricle presents no abnormality. Left auricle is much dilated and smooth; left ventricle contains a large quantity of currant-jelly clot completely filling the cavity. The aortic valves show each a broad line of recent vegetations below the edge and extending to the middle of the leaflets. The middle and posterior leaflets are joined 4 mm. beyond the point of insertion by a transverse band of vegetations. The intima of the aorta shows one patch of sclerosis; the caliber of the aorta is exceptionally small. The mitral valve shows recent vegetations along the edge on the auricular surface, and also on the extreme margin, and extending down one of the chordæ tendineæ. The valve and the chordæ tendineæ are thickened and shortened. The endocardium of the left auricle shows minute, yellowish, slightly rough, subendocardial spots. All vegetations, aortic and mitral, are short and low, like rows of short teeth. Thickness of left ventricle is 13 mm. The muscular substance is somewhat pale but firm. Weight of heart, 840 grams. The aortic valves are incompetent by the hydrostatic test.

Left lung: Consists of three lobes; pale; free from adhesions, except between upper and middle lobes. The lower lobe is somewhat edematous. Upper and middle lobes show considerable marginal emphysema. In lower lobe are a number of small infarcts; some on the anterolateral surface, and some on the surface directed toward upper lobe. These vary in size from a pin-head to a small pea, and are situated just within the pleura; all are pyramidal in shape and black in color. The bronchial glands are much enlarged, and on section are dark in color. Weight of lung, 280 grams.

Right lung: The three lobes are adherent by recent adhesions, and the lung is studded with numerous infarcts which are larger and greater in number than are those in the left lung. Infarcts are more numerous in the two lower than in the upper lobe. In center of lower lobe of right lung are tuberculous areas. One, about the size of a large pea, is surrounded by a not well-defined fibrous capsule. The bronchial glands are caseous at center. The entire lung is compressed, but still contains air, and exudes serum on pressure. Some pieces sink in water. Weight of right lung, 440 grams.

Spleen is pear-shaped, the pointed end directed downward; is firm, almost grits on section, shows prominent Malpighian bodies, no infarcts or tubercles. Weight, 190 grams.

Suprarenal capsules are small, dark, and soft.

Kidneys: Capsule strips fairly well; color pale; Malpighian bodies prominent by reflected light. Cortical striation is not well marked. Cortex seems wider than normal. Apices of pyramids are pale. Weight of left kidney, 140 grams; weight of right kidney, 170 grams.

Liver: Weight, 1540 grams. Transverse diameter 24½ cm., antero-posterior diameter 19 cm.; thickness 6 cm.; edges somewhat round. On section surface is mottled. There is considerable fatty infiltration, and also a yellowish staining of connective tissue apparently by bile.

Pancreas: Normal.

Mesenteric glands much enlarged, but not caseous. *May* 13, 1897.

IV. THE RESPIRATORY TRACT.

1. *Calcification of pleura, probably tuberculous in origin.*

Exhibited by J. A. Scott, M.D.

The specimen presented was secured by Dr. Romaine, of Lambertville, N. J.

The patient's history, which has but little bearing upon the specimen, is, briefly, as follows:

W. B. N., aged sixty-two years, a merchant by occupation; always had enjoyed good health; not exposed to cold in his business; his family history an exceptionally good one, in which there had been no phthisis or cardiac disease; habits as to alcohol and food good; with the exception of an attack of influenza when sixty years of age, had had no other illness. About July, 1894, he had a sudden and violent attack of dyspnea, which was relieved in a short time—a few hours; in October, 1894, a more severe dyspneic attack, and the third in the middle of November. Seen in consultation by Dr. J. H. Musser. Physical examination on December 6, 1894, showed a hypertrophied heart, with an accentuated pulmonary second sound and slight systolic rub; arterial tension increased; walls atheromatous; over the lungs, posteriorly, on the left, were lessened fremitus and bronchial breathing. Urine was 1027, acid; trace of albumin; ten or twelve hyaline casts. Patient was seen again on October 10, 1895; he then was in a mild uremic condition; the urine was highly albuminous; there was now a marked pulmonary diastolic murmur; evidence of right-sided dilatation; behind, at left base, were dulness, bronchial breathing, with bronchophony, and numerous râles. The patient died, apparently, from a myocarditis, right-sided dilated hypertrophy, and pulmonary congestion.

Upon autopsy, the heart was found much hypertrophied and dilated; the coronary arteries were reported as normal; there was no valvular disease; the lung on the left side was pressed up and carnified, and, extending from the fourth to the eighth dorsal rib, posteriorly, over the size of a man's hand, was a calcified area of pleura of varied thickness; this specimen is the sixth rib, with its attached pleura.

The kidneys and lung are now being sectioned.

The specimen here was removed from a man who had not, except during the last few months of his life, any pulmonary symptoms; no pleurisy, pneumonia, nor empyema; we know that this calcific infiltration is most apt to take place in tissues that are below par, or inflammatory areas and hemorrhagic infarcts, cysts, etc., especially after a chronic dry pleurisy with an organizing exudate, or any empyema, or, perhaps, a pleurisy of tuberculous origin. In this case, however, its origin is dubious, and gave rise to misleading physical signs; it was probably due to an old pleurisy, with calcific deposition.

Discussion.—Dr. Musser, in discussing the paper, said he had had an opportunity of seeing the man during life. He had a very decided hypertrophy of the heart, especially of the right heart. He also had passive congestion of the left lung and of the lower portion of the right lung. This congestion was most persistent, and he, the speaker, never had realized that a passive congestion, yielding physical signs of consolidation, could persist for such a long period of time. He saw the man quite often at intervals, the last time about six months before death. The physical signs presented were those of consolidation. He had lessened fremitus, posteriorly, due to the fact that the pleura was considerably thickened. During life a regurgitation was heard at the pulmonary orifice, as well as at the tricuspid, and he was quite sure that there was incompetence of both these valves, the tricuspid and the pulmonary. This condition is very rare.

Dr. Scott, in closing, said it might be possible to make a section of this calcified material and examine them for tubercle-bacilli. To him this would seem to be impossible, as it was so hard.

November 14, 1895.

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2. *Foreign bodies (coke) impacted in the bronchi.*

Presented by F. A. Packard, M.D.

The patient from whose body the specimen was removed was admitted to the men's medical ward of the Pennsylvania Hospital on July 24, 1895, and died two days after admission. He was a white man, aged forty-two years, and apparently a tramp. He would give no facts in regard to his family or past personal history, apparently because of a desire entirely to conceal his identity. The only statements that he

would vouchsafe were that he had been taken sick two days before admission, after a thorough wetting, and that he had spit a little blood at the onset. Further questioning caused the patient obstinately to refuse to speak. He was brought to the hospital in the ambulance from a railroad train. His condition from the outset was most serious. He was breathing rapidly and with great effort, the lips and skin were deeply cyanotic, and the pulse rapid and very compressible. His temperature was 102.8° on admission. Examination of the heart was entirely unsatisfactory, owing to the noisy respiration. Over both lungs there were loud, moist, bubbling râles, with scattered areas of finer adventitious sound, like large subcrepitant râles, which were barely audible through the larger sounds. Scattered over both lungs, especially posteriorly, there were small areas where the respiratory sounds had a rather tubular character. Over both lungs the percussion-note was in places decidedly impaired, and at both bases there was marked impairment, at no place amounting to decided dulness. The liver and spleen were of normal dimensions. The abdomen was slightly distended, but contained no demonstrable fluid. A diagnosis of bronchitis, with secondary bronchopneumonia, was made, and the patient was put upon carbonate of ammonium and tincture of digitalis, and a cotton-jacket with turpentine stupes every four hours was ordered. On the next day the patient's condition was in no wise improved, and strychnia, with atropin, was added to the treatment, while the cardiac stimulation of the day before was increased, and whiskey was given. Notwithstanding this the condition steadily grew worse, the dyspnea increased, in spite of the use of oxygen-inhalations, cyanosis became more marked, the sputum became blood-tinged, then bloody, and then mild delirium became more energetic. The patient died from cardiac and respiratory failure, struggling for breath until the last.

At autopsy there was found nothing of importance aside from the condition of the respiratory organs. Both lungs were deeply congested, and areas of collapse were numerous on both sides. On section there was found to be a large amount of bloody froth exuding from the parenchyma and from the cut bronchioles. Scattered throughout both lungs were areas of collapse and of consolidation. There was no abnormality of the larynx, trachea, or bronchi. On slitting the trachea there was found much injection of the whole mucous membrane. Just above the bifurcation of the trachea there were several small transverse ulcers seen, measuring from 3 to 5 mm. in their long axis by from 2 to 3 mm. in their shorter diameter. They extended down to the submucosa, had a punched-out appearance, and were covered by a rather

gangrenous-looking, thick, grayish membrane. In the right bronchus, just within its tracheal opening, there were found three hard bodies resembling coke (presented with the specimen). The largest of these was of a rusty-black color, irregular in shape, and measured 4 cm. in circumference, 1.5 cm. in its longest diameter. The medium stone measured 1 cm. in its longest diameter, the smallest 0.5 cm. These three bodies weighed together 19½ grains. At the point of the bronchus corresponding to the position of these bodies there were four rather gangrenous-looking ulcers, the largest measuring 11 mm. by 5 mm. in diameter. In the left bronchus there were two stones distinctly to be seen, with ulcers of the mucous membrane in their neighborhood, but the stones in this case were left in situ for preservation in the museum. After being in alcohol (70 per cent.) for twenty-four hours, measurements of the glottic chink were made and were found to equal 2 cm. by .75 cm. Careful search revealed no sign of perforation of the tracheal or bronchial wall from without.

The appearance of the foreign bodies was much like that of fragments of coke. That they might have been fragments of a calcareous tracheobronchial gland was the first thought suggested; but, as has been said, no perforation giving entrance to such fragments could be found. They were evidently not "lung-stones," both from their shape and character and from the fact that it would be unusual for several such bodies to be expectorated at one time, as they must have been in order to become impacted in the widening air-tubes. They must, therefore, have come from without. The manner and time of their entrance could not be at all determined from the history, as the patient evidently desired to keep his former movements secret. They can hardly be conceived of as entering through the glottis one by one, as the first one to enter would have quite effectually kept the glottis up to its work of protecting the orifice of the larynx, nor is it possible that the patient could have been forced to inhale so many foreign bodies if he were conscious. The only explanation that occurs to me is that the patient was a criminal tramp, that he was probably riding (as tramps frequently do) on the trucks of a train, that he fell asleep with his mouth widely open, and that a shower of coke from the road-bed was thrown in his face by the current of air produced by the train, and that particles were inhaled during inspiration. This sounds like a rather problematic explanation and of the Sherlock Holmes order; yet, on carefully thinking the case over, I can see no other method of accounting for the presence of so many foreign bodies of this size, shape, and character.

Dr. Stengel did not know if Dr. Packard had alluded to Dr. Osler's

case. The patient swallowed a peanut, and then coughed it up. She afterward developed pneumonia, and died.

Dr. Frank Savary Pearce remembered a case at the Presbyterian Hospital of something getting in through the larynx. The child had bronchial obstruction. On auscultation over the right lung a respiratory murmur could be heard, and then it would seem to be entirely absent. With these symptoms Dr. Wharton operated, and found half of a chestnut hull in the right bronchus at the junction of the trachea and the two bronchi. *December* 12, 1895.

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3. *The distribution and etiology of cardiac hydrothorax.*

Presented by J. Dutton Steele, M.D.

The literature concerning hydrothorax of cardiac disease, considered in relation to its etiology and to the location of the effusion, is small.

In general, the recognized authorities upon diseases of the heart and pleura dispose of the subject by the statement that the occurrence of effusion into the pleural sacs is part of a general dropsical tendency, occurring in the course of cardiac or renal disease, and due to diminished action of the heart and obstruction to the venous circulation. Of course, the fact is recognized that unilateral hydrothorax may be caused by the pressure of morbid growths upon the venous trunks. With a few exceptions, the statement is made that in heart-disease the effusion is always bilateral, though a few observers assert that there is apt to be more fluid on one side than upon the other, and a few writers mention the right side as oftener affected.

Wintrich,[1] in 1868, said that the effusion is almost always bilateral, but is usually greater on the right, and that it is unilateral only when there is an obliteration of one pleural sac. He, however, refers to one case (included in the list) in which there was a large right-sided unilateral effusion, without general edema, and with no lesions except a fatty heart and liver.

Fraentzel[2] states that the effusion is only unilateral when there is obliteration of one sac or some new growth interfering with the pleural circulation.

Wilson Fox[3] (1891) says that it is unusually bilateral, except when caused by the presence of tumors or other mechanical causes, but does not particularize.

Pepper,[4] in the section upon endocarditis in the *American Text Book of Medicine*, states that hydrothorax may occur in chronic valvular disease without general edema, and mentions three cases under observation, in which there was a greater amount of fluid on one side.

Strümpell[5] says, "It (the effusion) is usually bilateral, but is often unilateral, or at least larger on one side than upon the other.

Osler:[6] "It may, however, occur alone or with only slight edema of the feet. In heart affections one pleura is more commonly involved."

Villani[7] and Piazza-Martini[8] state that right unilateral hydrothorax is an early symptom of hepatic cirrhosis, and Villani adds that it often occurs in the early stage of venous congestion from failing compensation.

This subject has been particularly brought to my attention during the past year by several cases, seen in the Philadelphia Hospital, and by several conversations with Dr. Stengel upon the subject. The statement, made by him, that in his opinion effusion into the pleural sacs in the course of heart disease was more common and larger in amount upon the right side, together with an autopsy that seemed to throw light upon the subject, led to an analysis of a number of autopsies, and of a series of clinical observations, with the hope of determining reliable data upon the following points:

1. Whether, in the development of hydrothorax in the course of uncomplicated cases of heart-disease, both pleuræ were equally liable to contain effusion and in the same amount.

2. If such was not the case, which side was oftenest affected.

3. Whether the effusion was ever entirely unilateral, and, if so, on which side.

4. Whether hydrothorax showed a tendency to develop before general dropsy occurred.

In the analysis of the autopsy-notes only those cases were taken in which the myocardium had undergone sufficient hypertrophy to increase the weight of the heart to 400 grams in the adult male and to 370 grams in the adult female, or in those in which it had undergone chronic degenerative changes leading to dilatation of the heart-chambers. The object of such requirements was to exclude cases of dilatation secondary to some acute disease, and also dropsies occurring in the last hours of life, and to limit the data to cases of effusion due solely to uncomplicated cardiac disease.

Further, cases in which concomitant conditions occurred that might cause effusion were excluded. Such are acute pleurisy, aneurysm, morbid growths of the pleura or mediastinum, or enlargements of the mediastinal glands.

The following analysis was based upon the notes of 1652 autopsies reported in the *Middlesex Hospital Reports* for the six years ending 1896, and upon those of 775 autopsies made at the Philadelphia Hospital from March, 1893, to September, 1896. The total number of autopsies was 2427.

Total number of cardiac cases in which the requirements were fulfilled	173
Total number of cases of hydrothorax	75
Bilateral equal hydrothorax	22, or 30 per cent.
Bilateral unequal hydrothorax	40, or 53 " "
Unilateral hydrothorax	13, or 17 " "
Bilateral unequal hydrothorax, right greater	31, or 77 " "
Bilateral unequal hydrothorax, left greater	9, or 23 " "
Unilateral hydrothorax, right	10
Unilateral hydrothorax, left	3

It will be seen that in 70 per cent. of the cases of hydrothorax the effusion is unequal. In unequal bilateral effusions the effusion was greater upon the right in thirty-one instances from a total of forty (77 per cent.).

The effusion may be unilateral, and then is more than three times as often found upon the right.

These percentages are too high to be explained by accidental circumstances, as decubitus, and even if a certain amount of the transudation took place during the last hours of life, there is no reason why it should be unequally distributed.

In the thirteen cases of unilateral effusion two cases showed slight edema of the lower extremities and one ascites. In the remaining ten cases no mention is made of the presence or absence of edema. This negative evidence seems to be in favor of its absence in at least a proportion of the cases.

Of the forty cases of unequal bilateral effusion four are recorded as having general anasarca, five as having edema of the lower extremeties, and two are reported as free from all dropsy. In the first of the last mentioned the right pleura contained forty ounces and the left four ounces. In the other instance the right pleura contained twelve ounces and the left two ounces, and were practically unilateral.

Of the twenty-two cases of equal bilateral effusion eight had general anasarca, and one edema of the legs. Nine were entirely free from dropsy. In thirteen cases no mention is made of the condition in this respect. These figures are incomplete, owing to lack of positive evidence as to the absence of edema, but it is worthy of remark that the tendency to general anasarca is greatly increased in those cases in which the

pleural effusion is equal, and in the unequal cases less than one-sixth of the total are recorded as showing any edema.

In presenting the following series of cases, my thanks are due to Dr. F. A. Packard for the notes of Case I., and to Dr. Alfred Stengel for the opportunity of observing Cases II., III., and IV. in his wards of the Philadelphia Hospital during the summer of 1896.

Case I.—P. M., male; white; born in Ireland; aged fifty-six years; occupation, laborer. He had been a very healthy man up to the spring of 1892, when he first began to suffer from dyspnea on exertion. He was under medical treatment until April, 1893, when he came into the Philadelphia Hospital. On admission he was suffering from severe orthopnea and weak and irregular pulse, with physical signs practically the same as recorded below. His right side was tapped soon after admission and a large amount of clear fluid was withdrawn, with great relief. He had no fever, no pain, and no cough. During the following year he remained in the hospital, and in this time fluid was drawn five times from the right pleural sac and once from the left. During all this time he had very slight edema of the feet, but orthopnea was often marked.

September 7, 1894. The following note was made: Orthopnea is frequent, but edema of feet rarely occurs. Examination: No bulging of precordia. Area of impulse not enlarged. Apex-beat in the fifth interspace, one-half inch to left of midclavicular line. There is a thrill in the arteries of the neck. Cardiac dulness extends from the lower border of the third rib above. The left margin is one half inch outside the midclavicular line. The right margin is the left border of the sternum. There is a high-pitched harsh systolic murmur at the apex that is short in duration, and is well transmitted to the axilla. There is also a short, lower-pitched, presystolic murmur heard best just above the apex. In the aortic area there is a rough, short systolic murmur transmitted into the arteries of the neck, and a very short, rough diastolic murmur as well, transmitted down the sternum. Pulmonary and tricuspid areas present no modification of the normal heart-sounds. There is bilateral pleural effusion that reaches on the right to the fifth rib posteriorly, and on the left to the seventh rib posteriorly. He suffers considerably from orthopnea. Respirations 40. Patient is confined to bed. From this time on the patient grew stronger and was able to be around the wards. His heart's action became much more regular, the amount of urine increased, and the pleural effusion gradually diminished. Upon September 22 the fluid reached the eighth rib posteriorly on the right, and had entirely disappeared on the left.

October 26, 1894. Patient has shown increasing weakness for several

days, with attacks of cyanosis and dyspnea. The fluid has been rapidly increasing in the right pleural sac in the same period, and now reaches to the spine of the scapula. He was tapped on the next day, and seventy-two ounces of clear yellow fluid was withdrawn. This fluid had a specific gravity of 1019, contained a moderate amount of albumin, and, microscopically, many red blood-cells and a very few leukocytes.

28*th*. Patient gradually grew weaker and died suddenly to-day. It is to be regretted that no autopsy was obtainable.

The feature of this case is the presence of bilateral hydrothorax, greater upon the right, that increased and diminished as the heart lost or gained strength. The condition of the heart may reasonably be assumed to be aortic, and mitral stenosis, failure of the myocardium, and dilatation following hypertrophy. There is no indication of pleurisy, aneurysm, or mediastinal tumor, and there are no symptoms pointing to hepatic disturbances. The hydrothorax preceded the appearance of edema.

Case II.—A. M.; white; male; native of Italy; aged thirty-one years; married. Six years ago he was ill for some time with a severe pain in the right side of his chest. A physician applied leeches, with relief. Syphilitic infection one year ago. He uses alcohol in moderation. Six months ago he began to suffer from a dry cough, accompanied by much pain over the right side of the thorax. He was in a hospital for four months, and stated that while there his chest had been tapped, presumably upon the right. One month before admission his legs, feet, and, later, the scrotum commenced to swell, and a short time before entering the hospital he noticed some enlargement of the abdomen. He suffered severe pain in the right thoracic region during this time, and the slightest exertion excited much dyspnea.

Examination. Heart, fetal rhythm, and occasionally a faint systolic whiff at the apex, but usually no murmurs can be made out. There is no murmur at the aortic or tricuspid valves. The second sound is accentuated in the aortic area. The heart is much enlarged to the right, and moderately so to the left.

Thorax. Effusion into the right pleural sac, reaching to the third rib, anteriorly. A few moist râles are heard above the effusion, anteriorly and posteriorly. The breathing is harsh on the left side, but this pleural sac is free. The spleen and liver are enlarged, and there is considerable tenderness over both organs. There is moderate edema of the legs and scrotum and abdominal and thoracic walls. There is a small amount of ascites. A small syringeful of clear yellow fluid was withdrawn from the right side.

For the two weeks following admission his temperature ranged from 97.6° to 101° F., and after this it gradually fell to normal. During the next month the edema disappeared, the temperature fell to normal, and the patient recovered sufficiently to be out of bed. As the heart gained in strength a low-pitched mitral regurgitant murmur developed.

He gradually became melancholic, but when last heard from his general somatic condition was much improved and he was free from all dropsy. The probabilities are that this patient, six years before, experienced an acute attack of pleurisy on the right side.

The sequence of events was myocarditis from some cause, probably alcoholism, acute dilatation of the heart with right unilateral hydrothorax, preceding general edema. The formation of the effusion was accompanied by enlargement and tenderness of the liver, ascites, and some pain in the right thoracic region. The effusion disappeared as the heart gained strength.

Case III.—C. U., chair-caner; aged twenty-nine years; has not worked at his trade very continuously, and has done much hard manual labor. Has lived in Philadelphia all his life.

Family history. His father is alive, and has suffered much with rheumatism. Mother died of some septic infection, and had pus in her knee at time of death. One brother died of consumption.

The patient had measles in childhood, but never had scarlet fever or diphtheria. Has never had rheumatism. He has used alcohol to great excess for three years. Denies any venereal history. About a year ago, following a debauch, he began to suffer from dyspnea and vertigo. In the past year has been in several hospitals for severe attacks of the above symptoms. He recovers after a period of rest, and is comparatively comfortable until physical exertion brings on the trouble anew.

Admitted to Philadelphia Hospital July 20, 1896. Examination July 29th. The patient is anemic, and his color suggests nephritis. The veins of the neck are enlarged, but do not pulsate. Pupils are equal. There is no tracheal tugging.

The apex-beat is seen in the fifth interspace, one centimeter outside of the midclavicular line. It is somewhat diffused. Another impulse is seen in the fourth interspace in the midclavicular line, to the left of the sternum; also somewhat diffuse. Palpation shows a faint systolic thrill over the apex-beat. Percussion shows the heart to be pushed somewhat to the left. The left border by auscultatory percussion is 3.2 centimeters to the left of the midclavicular line, at the left of the fifth rib. The upper border corresponds to the upper edge of the third rib; the lower border to the lower edge of the sixth rib; the right to the mid-

sternal line. There is a faint systolic murmur at the apex, which is rough and galloping and suggests the possibility of its being presystolic in time. At the tricuspid area, the first sound is soft and murmurous, and is of a distinctly different quality from that heard at the apex. No distinct murmur can, however, be made out. There is no murmur at the aortic area. The second sound in the pulmonic area is accentuated. The right side of the thorax has decidedly less movement than the left. There is dulness on the right side up to the angle of the scapula, posteriorly, and to the upper border of the fourth rib, anteriorly. The line of dulness changes when the patient changes his position. In this area the breath-sounds are suppressed, and the vocal resonance and fremitus are absent. Along the upper border of dulness, faint egophony can be heard. The aorta shows no abnormal pulsation, and there is no area of dulness suggesting an aneurysm.

The breath-sounds are somewhat harsh in both lungs, especially marked above the effusion in the right. A few dry râles are heard over both sides.

Liver dulness commences at sixth rib and extends downward 3.5 cm. in the midclavicular line and 4.5 cm. in the midaxillary line. Posteriorly it commences at the ninth rib and extends downward 9 cm. It is therefore distinctly reduced in size. The area of splenic dulness is somewhat larger than normal.

The patient gained strength and the dyspnea gradually left him. As his heart's action became stronger the effusion lessened, and within two weeks from the time of admission had entirely disappeared.

The patient grew strong enough to be about the wards, and remained in the same condition until August 20, 1896, when he dropped dead while walking across the ward. No autopsy.

In this case the sequence of events was probably as follows: Alcoholic myocarditis; acute dilatation, particularly affecting the right chambers; right-sided unilateral hydrothorax, diminishing in amount as compensatory hypertrophy occurred. There was absence of all signs of mediastinal tumor and hepatic enlargement, of tenderness, and of ascites. There was but very slight subcutaneous edema.

Case IV.—M. S., German, miller, widower, fifty-three years of age. The patient has no friends, and speaks an unintelligible dialect, and no history can be obtained.

He was admitted to the Philadelphia Hospital on June 22, 1896. He was suffering then with great dyspnea, cyanosis, but there was no edema of the extremities. The heart was irregular and rapid. There was an impulse in the anterior axillary line on the left. The heart was

so irregular that no murmur could be made out. There was dulness over the whole of the right side, with suppressed heart-sounds, and absence of vocal resonance and fremitus. The liver extended to the level of the umbilicus in the midclavicular line, and there was some tenderness over it. The urine showed no albumin. Its specific gravity was 1025. The right pleural sac was tapped and sixty-three ounces of a clear fluid was obtained, the specific gravity of which was 1019. The patient was much relieved, and the heart returned almost to its normal position. The apex was then in the fifth interspace in the left midclavicular line. He improved slowly and was fairly comfortable for several weeks, though the heart's action continued somewhat irregular, and the sounds indicated weakness of the heart-muscle.

The right pleural sac began to fill up slowly soon after the aspiration, and upon July 11 the right sac contained considerable fluid. The heart is still very weak, and a very faint, rough, systolic murmur can sometimes be heard at the apex. The liver still reaches to the level of the umbilicus. Diaphragmatic movement is absent on the right side.

Urine, specific gravity, 1018; no albumin; forty ounces in twenty-four hours. The amount of urine varied from this date to time of death from twenty-five to thirty ounces in the twenty-four hours. It never contained albumin.

The patient, after the last note was made, began to experience attacks of dyspnea and cyanosis, and died in such an attack July 20, 1896.

Autopsy, twenty-hours after death. The body of a fairly well-preserved man. There is slight edema of legs, ankles, and scrotum.

The abdominal cavity contains about two hundred and eighty cubic centimeters of clear, serous fluid. The diaphragm is pushed down so that on the right the upper border of the liver is three centimeters above the costal margin in the midclavicular line, and nine centimeters in the medial line.

The heart is pushed downward. The apex is in the midclavicular line, under the seventh rib. The right auricular is much distended, and projects six centimeters to the right of the median line at the level of the root of the right lung.

The dilated right auricle presses directly on the root of the lung. The bronchus shows no evidence of compression. Veins of right pleura not congested. The vena cava shares in the dilatation of the right auricle. The thoracic duct is not dilated.

The heart is much dilated by liquid blood. The wall of the left auricle measures 4 millimeters in thickness, and that of the right auricle the same. Left ventricular wall is 26 millimeters in thickness;

right ventricle, 6 millimeters. Weight of heart, 640 grams. The myocardium is pale, with several areas of increased connective tissue scattered through it. The muscle is friable and soft, but there is no marked fatty change. The endocardium of the lower third of the left ventricle is covered by marantic coagula, which, in places, are firmly adherent to the endocardium, and are entangled between the fibers of the columnæ carneæ. There are a few spots of fibrous thickening upon the aortic valves. The free edge of the posterior leaflet of the mitral is thickened by fibrous change. The tip of the papillary muscle of the left ventricle is much thickened by increase of fibrous tissue. The right pleural sac contains 1600 cubic centimeters of clear, blood-stained fluid. The visceral layer of the pleura is somewhat thickened, but there is no sign of an inflammatory process, and the fluid contains no fibrin. The left pleura contains 300 cubic centimeters of clear, blood-stained fluid.

The left lung is intensely congested and edematous, especially at the bases, posteriorly, but it is everywhere crepitant. The right lung is small and compressed, but shows no pathologic condition, except congestion and edema.

Spleen is small, and measures 9 by 7 by 3 centimeters. The capsule is thickened and wrinkled. The organ is cyanotic and indurated. In the lower portion of the organ is a small, pyramidally-shaped scar that extends deeply into the organ. The adrenals are normal.

The kidneys are very small. The capsule strips with great difficulty. The surface of the cortex shows many cysts, and is scarred. The cortex is very much contracted. On section, the surface of the section is so much distorted by fibrous change that the pyramids and cortex can scarcely be distinguished from each other. Left kidney weighs 90 grams. Right kidney weighs 110 grams.

The pelves of the kidneys and ureters of both sides show small, herpetic vesicles, filled with clear fluid. These vesicles measure 1 millimeter in diameter and are not easily broken. The ureters and bladder are otherwise normal. The pancreas is indurated. The liver is slightly smaller than normal. There are a few adhesions between the liver and the diaphragm evidently due to an old perihepatitis. On section, the central veins are engorged. The consistency of the liver-tissue is not increased, and it is friable and distinctly fatty in spots. The glands of the mediastinum are not enlarged or diseased. Nothing else of pathologic interest is found in the body.

In this case there were arteriocapillary sclerosis, chronic fibrous myocarditis, hypertrophy, and dilatation, especially affecting the right

auricle. There was hydrothorax greater on the right side, increasing as the heart lost strength. There was no fever, pain, or sign of inflammation in the pleura or abdomen. Slight ascites and edema existed, consistent with commencing tendency to general dropsy, but the thoracic effusion preceded the appearance of the subcutaneous edema.

Microscopic examination of the heart-muscle and kidneys confirmed the diagnosis of fibrous myocarditis and interstitial nephritis.

Case V.—(Reported by Wintrich.) A small, fat, thick-necked man, a waiter by occupation, and aged thirty-six years, was admitted to a hospital in Berlin. He was cyanosed and almost pulseless. The veins of his neck were over-filled; the extremities were mottled by blue spots, and the respirations were stertorous. He died in forty-eight hours, without a thorough examination, and before a history could be obtained.

Autopsy. There was no edema of any portion of the body, except, perhaps, that the subcutaneous fat showed an excessive amount of moisture. The right pleura contained a very great amount of clear, yellow fluid. The pleura showed no sign of inflammation whatever. There was no neoplastic growth anywhere in the body. There was no fluid in the left pleural sac nor in any of the other serous cavities. The right lung was compressed, but otherwise showed no pathologic condition. The left lung was normal.

The heart was not greatly infiltrated with fat, but the muscle was weak, soft, and dull in color. The blood in the heart-cavities was dark, cherry-red, and fluid, and of the same character as that found in the rest of the vascular system. The valves were normal. The vena cava descendens was very much engorged and dilated.

There were no pathologic changes in the abdomen. The liver was fatty. The spleen was enlarged, hard, and firm. Otherwise there was nothing abnormal found in the body. Wintrich remarks that the above is a very instructive case of unilateral hydrothorax, for which no cause can be found other than chronic myocarditis from alcoholism. No explanation for the conditions found can be given, except a dilated right auricle. There was no inflammatory change about the liver or ascites. There was practically no subcutaneous edema.

Case VI.—(Reported by Guitéras.[9]) Male; aged twenty-seven years; occupation, sailor. He was always in good health, except an attack of acute articular rheumatism at twenty-two years of age, followed by no apparent heart-lesion. Soon after this he contracted syphilis, and came into the hospital twice for the secondary manifestations. Three years before admission he began to suffer from dizziness, dyspnea, and palpi-

tation. During the present year he was twice under treatment for edema and other cardiac symptoms.

Examination. Emaciation and slight jaundice. The face and extremities are congested and the veins of the neck are swollen and pulsating. An ophthalmoscopic examination shows pulsations of the retinal veins. There is marked epigastric pulsation, and a diffuse apex-beat in the fourth and fifth interspace in the midclavicular line. There is a blowing mitral systolic murmur transmitted to the axilla and to the right toward the ensiform (tricuspid systolic murmur?). The second sound is reduplicated in the aortic area.

Autopsy. Lungs edematous. No adhesions in the pleural sac. There is considerable passive effusion in the right pleural cavity.

Pericardium is closely adherent to the entire surface of the heart.

The left ventricle is somewhat dilated. Its walls are of normal thickness. There is some thickening of the auricular walls. The mitral valve is stenosed so as to form a button-hole orifice. The right ventricle is slightly hypertrophied. The right auricle is dilated and its wall is thinned. The cusps of the tricuspid valve are thickened and shortened. There is some stenosis of the aortic valves. The myocardium is flabby and evidently fatty. The heart weighs 570 grams. The liver and kidneys are cyanotic and fatty.

The sequence of events was probably rheumatic valvulitis, compensatory hypertrophy, gradual degeneration of the myocardium, and dilatation, especially affecting the right auricle. Considerable right unilateral hydrothorax with but slight general edema. No signs of inflammatory change in the pleura or serosa hepatica. No enlargement or tenderness of the liver.

Piazza-Martini reports several cases of right unilateral hydrothorax occurring in atrophic cirrhosis of the liver, and offers as an explanation for the condition the fact that the esophageal veins are engorged by the attempt to establish a collateral circulation between the obstructed portal system and the general venous system. These engorged veins press upon the vena azygos and interfere with the return of blood from the right pleura, and hence the effusion. His theory was upheld by Lizzato[10] and Cardanelli.[11]

Villani also stated that he had observed the presence of a considerable serous exudate in the right thoracic cavity as a constant symptom of atrophic hepatic cirrhosis. He reports nine cases in which this occurred. The effusion occurred without fever, stabbing pain on respiration, or painful cough, and he therefore excluded acute pleurisy as a causal factor. He opposes Piazzi-Martini's theory of the origin of the

fluid, and gives as an explanation that the pressure upon the serous covering of the liver in the first stages of interstitial inflammation, as well as its participation in the inflammatory process, causes a low grade inflammation of this coat and some effusion into the peritoneal cavity. The morbid process thus spreads through the diaphragm by continuity to the pleura. There, also, low-grade inflammatory changes are set up and an exudate is formed in the pleural sac.

Villani goes still further, and has observed the presence of right-sided hydrothorax in cardiac disease, uncomplicated by interstitial change in the liver. He gives as an explanation the fact that in failing compensation of the heart the liver is the first structure to feel the venous obstruction, the peritoneum hepaticum is stretched and inflamed, and the hydrothorax developed in the sequence given.

Villani's theory, while plausible in certain cases, is not comprehensive. Thus it does not explain how in some instances the effusion is entirely left-sided. It is hard to conceive how an effusion, occurring in the manner that he states, could reach the dimensions seen in some of the cases in the clinical list without some fibrinous exudate and subjective symptoms. In Case II. such symptoms were present. There were enlargement of the liver, tenderness on pressure in the hepatic region, considerable pain over the right side of the thorax, and some fever.

In the autopsy upon Case IV. considerable perihepatitis and some ascites were found, but the fluid was entirely free from fibrin, and the specific gravity and percentage of albumin were too low to be consistent with an inflammatory origin for the effusion.

Cases I. and III., however, showed no ascites, and there was no pain or fever. Here, then, are two cases in which Villani's theory is insufficient to account for the unilateral effusion that was present.

The amount of effusion in Cases I. and III. evidently depended upon the condition of the heart. It increased when the heart weakened, and decreased as the heart gained strength. In Case I. it almost disappeared from the right pleura, and was entirely gone from the left when the patient recovered sufficiently to be about the ward. In Case III. the right-sided effusion disappeared as the heart grew stronger.

In Case IV. the effusion increased as the heart failed, and at the autopsy the liver, though cyanotic, was not markedly so, and was not enlarged. In none of these cases was there hepatic tenderness while the fluid was increasing.

If, however, a low-grade inflammation was once established in the diaphragmatic pleura on the right, it is quite conceivable that a venous engorgement of this part would tend to prevent its resolution, and thus

the effusion might be maintained. But if the effusion was due to an inflammatory process secondary to similar changes in the liver, it is hard to see how the fluid could increase without an increase in the enlargement and tenderness of the liver, a condition entirely absent in all of my cases. It is worthy of note in this connection that in the sixty cases of right unequal hydrothorax there were but six cases of abdominal effusion without general dropsy; a very small proportion if the primary cause of the excess of fluid upon the right side was due to an inflammatory process commencing in the serosa hepatica.

In 1838 King[12] reported five cases of mitral stenosis in which the left bronchus was compressed to a considerable degree by a dilated left auricle. In two of these cases there was unilateral left-sided hydrothorax. In one of the remaining cases there was obliteration of both sacs, and in a fourth an acute pleurisy. In the fifth case the bronchus was but slightly constricted, and the pleural sacs were empty.

Friedrich[13] also reports a case of mitral stenosis with a similar condition, but does not mention the condition of the pleura. When it is considered that in the cases of right-sided hydrothorax given there was evidence of dilatation of the right heart, and in King's cases half showed left-sided effusion, the idea strongly suggests itself that pressure exerted by a dilated heart may be an important factor in the causation of unequal pleural effusion. In King's cases pressure was exerted directly upon the root of the left lung. The right auricle is in closer relation with the root of the right lung than the left auricle is to that of the left. In Case IV. the dilated right auricle was found to press directly upon the vessels and bronchi of the right lung, although the bronchus itself showed no evidences of compression.

The relations of the roots of the lung are thus given by Quain:[14] "The root of the right lung lies behind the superior vena cava and part of the right auricle, and below the azygos vein, which arches over it to enter the superior cava. That of the left lung passes below the arch of the aorta and in front of the descending aorta"— that is, the right auricle lies in front and in intimate relation with the root of the right lung, and the left auricle below and also in intimate relation with that of the left lung. I have confirmed this statement by observation upon a series of cases in the autopsy-room.

The venous circulation of the pleural sacs is in the largest part accomplished by the intercostal and bronchial veins, while the thymic, pericardial, and internal mammary play a very unimportant part in it. The root of each lung is composed of the bronchus, the large blood-vessels, the lymphatics of the lungs, and the bronchial vessels (Quain).

The lymphatics of the lung form a close network under the visceral layer of the pleura, and unite in large trunks that pass out to the bronchial glands along the root of the lung.

Pressure upon the roots of the lungs, if not sufficient to compress the larger vessels, might, however, seriously interfere with the circulation of the lymphatic vessels and bronchial veins. The disturbance in the bronchial circulation would probably not be marked because of their anastomosis with the pulmonary circulation. Pressure upon the root of the right lung would also compress the vena azygos, which passes directly behind it. This vessel receives all the intercostal veins from the right side, the lower ones directly and the three upper through the right superior intercostal veins. It also receives as tributary the vena azygos minor, which receives the intercostal veins from the lower three or four intercostal spaces on the left. The left intercostal veins, from the fourth to the seventh or eighth interspaces inclusive, empty into the left upper azygos vein, which in turn opens into the vena azygos major directly or in common with the vena azygos minor. This left upper azygos vein communicates above with the superior intercostal vein of the left side, which opens into the left innominate vein and receives the intercostals of the three upper spaces. The left bronchial vein empties into the vena azygos minor, the right into the vena azygos.

Pressure, then, upon the vena azygos, such as could be occasioned by a dilated right auricle, would interfere with the return circulation of the right pleural sac, and to some extent with that of the left, but the communication of the left upper azygos vein with the left innominate would relieve to some extent the obstruction upon the left side.

To formulate the results of pressure exerted by a dilated right auricle and vena cava upon the root of the lung:

(1) Obstruction to the venous circulation of the right pleural sac through the vena azygos and its tributaries, and to a less extent through the bronchial veins.

(2) Obstruction to the lymphatic vessels supplying the visceral layer of the pleura.

Pressure of a dilated left auricle would cause:

(1) Obstruction to the circulation of the bronchial veins and lymphatics of the left lung.

(2) Obstruction in the left superior intercostal vein and vena azygos minor, which lie behind this auricle in the posterior mediastinum.

In sixteen of the thirty-five cases of right unequal bilateral hydrothorax dilatation of the right auricle is especially mentioned. And in

two of the five cases of unequal left hydrothorax the left is stated to be particularly dilated.

The early occurrence of hydrothorax in cardiac disease and its tendency to affect the right side earlier than the left is indicated in the five cases of the clinical series. The great inequality of the effusion in favor of the right side is shown in the series of autopsies. The possibility of pressure by a dilated right auricle upon the root of the right lung, and consequent interference with the return circulation of the pleural sacs with the production of a passive effusion, has also been shown. When these facts are coupled with the tendency of the right auricle to be the chamber most liable to be dilated in failing compensation of the heart, the connection seems to be more than a coincidence and appears to be a reasonable explanation.

In a similar manner the cases of left unilateral effusion, and unequal bilateral effusion greater upon the left, may be explained by interference with the return circulation of the left pleura by pressure upon the root of the left lung and the left superior intercostal vein, a condition which occurred in two of King's cases.

While it has been repeatedly proven that temporary obstruction to the venous circulation is insufficient to cause edema in the part affected, yet all observers unite in the statement that long-continued obstruction will cause local edema, whatever be their theory concerning the pathology of dropsy. Lazarus-Barlow[15] and Starling[16] unite in the opinion that an impoverished condition of the blood-plasma, and consequent malnutrition of the tissues, are most important factors in the causation of dropsy.

The equal bilateral pleural effusions, then, may be explained by the supposition that the malnutrition of the body tissues in general was considerable before the dilatation of the auricles was sufficient to exert localized pressure, and the tendency to general dropsy occurred as soon as the failing heart offered the slightest obstruction to the venous circulation, affecting the pleura as the other serous cavities.

Conclusions.—During the failure of the heart-muscle in uncomplicated cases of chronic heart disease, passive effusion into the pleural sacs may occur before there is any tendency to dropsy elsewhere in the body.

In the great majority of the list of cases of hydrothorax of cardiac origin the effusion was bilateral, but it was usually (70 per cent.) unequal in distribution, and three-fourths (77 per cent.) of the unequal effusions were greater upon the right side.

The hydrothorax may be entirely unilateral, and five-sixths of these cases were right-sided.

The explanation is furnished by the fact that when either auricle is

dilated it obstructs, by its pressure, the venous and lymphatic circulation of the pleura of its own side, and thus causes effusion into its sac. This usually occurs before the interference with the systematic venous circulation is sufficient to cause general dropsy. The position of the right auricle in relation to the other chambers of the heart renders it most liable to dilatation, and hence effusion occurs oftener upon the right in unilateral cases, and in bilateral is greater upon this side.

Equal bilateral hydrothorax is explained by a cachectic condition of the blood and tissues, and in the cases analyzed was very much oftener associated with general dropsy than the unequal form.

The presence of low-grade inflammation in the pleura that has been excited by an old pleuritis or by a similar condition in the serous covering of the liver, caused by the stretching subsequent to its enlargement from congestion, may, and probably does, account for a certain proportion of cases, but in the great majority of instances this explanation is insufficient.

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January 14, 1897.

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4. *Gangrene of lung.*

Exhibited by M. H. Fussell, M.D.

John McC., aged forty years, grave-digger; one brother died of tuberculosis; anything further concerning his family history cannot be ascertained. As to his previous history nothing is known other than the fact that he has always been a very free user of alcoholic beverages, especially beer. He has undergone a great deal of exposure, such as sleeping out of doors and in unoccupied and unheated buildings. When

seen on the afternoon before being brought to hospital he was very much intoxicated. That night, about twelve o'clock, an officer heard groans in an old church, and on entering found the man lying on the floor, apparently drunk. He was taken to the station-house, where he was put in a cell for the night. On the way to the station-house he conversed with the officer. Toward morning, as the night watchman made his rounds, he found the man lying on the floor of his cell in an unconscious condition. His face on one side was slightly contused as the result of falling off the bench in the cell. He could not be aroused, and was at once sent to the hospital.

When first seen at hospital he was unconscious, and could not be aroused. His pupils were widely dilated, and could not be made to react by light. His breathing was shallow, and his breath bore the odor of alcohol. Urine and feces were passed involuntarily. Later on he had several convulsions, lasting from one-half to five minutes. After this a specimen of urine was obtained by catheter, and it was found to contain both albumin and casts. In about forty-eight hours he began to regain consciousness, and in the course of a week's time was quite rational. The urine, however, continued to be albuminous and contained casts.

Within the next few days he developed a severe cough and expectorated quite freely. Sputa examined, but no tubercle-bacilli found. About this time he began to complain of severe pain on the right side, at the lower border of the liver, though it was not constant, and he ceased to complain of it in two or three days. From the beginning of the cough his temperature went up. He gradually grew weaker until eight days later, when he died.

Autopsy. Kidney. Cortical substance greatly in excess of medullary. Left suprarenal capsule greatly enlarged; one-quarter of kidney softened.

Right suprarenal capsule very greatly enlarged; capsule congested and seat of little tubercular mass. Kidney itself is "large, white."

Right lung. Upper lobe—the seat of extensive tuberculous infiltration. Middle lobe—a few scattered tuberculous masses. Lower lobe—the seat of an extensive gangrenous cavity, occupying entire lower half of lobe.

Left lung. Upper lobe full of cheesy masses and miliary tubercles. Tip of lower lobe, tuberculous cavity.

Heart. Hypertrophied. *April* 8, 1897.

V. THE GENITO-URINARY SYSTEM.

1. *Tuberculosis of the kidney.*

Exhibited by M. Howard Fussell, M.D., and J. A. Johnston, M.D.

W. R., aged twenty-seven, a mover, referred by Dr. John Weaver, of Norristown, to the Medical Dispensary, Norristown Hospital, May 22, 1895.

Family-history entirely negative. Had measles in infancy, influenza in 1890. In November, 1894, while lifting a heavy weight, fell and struck his back, and since then has had pain, more or less constantly, in right lumbar region. Pain is greater in damp weather. Voids urine three or four times during day, and occasionally has to rise at night. Never any hematuria.

Physical examination. Lungs negative; second aortic sound of heart much accentuated. Prominence in right lumbar region, pressure upon which occasioned great pain. Examination of urine on this date: Reaction, strongly acid; specific gravity, 1031; albumin, one-tenth by bulk. Large quantity of pus and many tubercle-bacilli were found. June 3, 1895, examination of lungs revealed increased tactile fremitus, high-pitched percussion note, with a prolonged expiration at right apex. Prominence in right lumbar region disappeared. Fewer bacilli found in urine. Patient became too ill to return to dispensary, but his condition was reported by Dr. Weaver, through whose courtesy we were enabled to make the autopsy.

Autopsy made forty-eight hours after death. Body that of a much emaciated man. Rigor mortis well marked. The abdominal cavity filled with a reddish-brown fluid, presumably embalming fluid. No evidences of peritonitis. On opening the thorax the right lung was found to be markedly congested. Pericardium contained a small amount of fluid. On opening the heart the cavities were found to be filled with fluid blood. Right side contained a chicken-fat clot; mitral valve was free. Great dilatation of tricuspid orifice, and, in fact, of the whole right heart. Caseation of the posterior bronchial glands.

Right lung was crepitant, except at extreme apex, where there was a point of consolidation. The surface of the pleura was studded with

numerous miliary tubercles, especially between the lobes. On section of the apex, a very recent point of infiltration was found. The whole lung was studded throughout with miliary tubercles.

Left lung. Slight infiltration at apex, and studded throughout with miliary tubercles.

Liver. A few miliary bodies scattered over surface of liver, both upper and lower. On right lobe especially these tubercles are quite extensive, apparently extending below the surface.

Pancreas was normal.

Spleen. Capsule covered with recent adhesions. On section the body is studded with miliary tubercles.

Bladder contained a large amount of purulent fluid, and tore very easily; mucous membrane pale, and free from ulceration or hypertrophy.

Left kidney. Had a curious lobulated appearance. Kidney itself pale in color. The cortical and medullary portions have a normal relation to each other. A few small miliary tubercles found at the apices of the pyramids, but none had undergone softening.

Right kidney. Capsule is apparently thickened and somewhat adherent. Kidney substance is pale. In the point of the uppermost papilla there is a small softened area, and numerous miliary tubercles are found scattered through the pelvis.

The case is a rather interesting one, in that there were so few physical signs present. *November* 14, 1895.

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2. *Primary renal tuberculosis, with involvement of the adrenals and the seminal vesicles.*

Exhibited by Alfred Stengel, M.D.

The patient, a young man suffering with pulmonary phthisis, presented symptoms, toward the end of life, indicating intestinal involvement and rapidly succumbed.

At autopsy, tuberculosis of the lungs was determined and the intestines presented a few small ulcerations, with small miliary tubercles on the serous surface. The lesions of interest, however, were found in the genito-urinary apparatus. The left kidney presented a solitary nodule about the size of a walnut, occupying the cortex of the kidney and producing a distinct elevation of its surface. On section, this was found to be cheesy within and was sharply delimited from the surround-

ing renal structure. The pyramids of the kidney were healthy in appearance. There were no other nodules. The right kidney was apparently normal. The pelvis of both organs was normal, as were also the ureters and bladder. Both seminal vesicles were involved, presenting, near the end, small nodules, about the size of a cherry, springing from the wall, and having the same structure as the nodule in the kidney. The right contained a certain amount of granular, cheesy material, and was slightly encrusted with calcareous matter near the prostatic end, and the prostate at this situation was involved for a short distance around the duct. The testicles were normal. The suprarenal capsules of both sides were enlarged and the medulla converted into a caseous tissue. The organs were free from adhesions to the surrounding structures.

The title given to this paper is entirely inappropriate in a certain sense—it is not contended that the renal lesion in this case was strictly a primary one. It probably resulted from hematogenous infection secondarily to the pulmonary condition, but in form and situation it resembles the cases of so-called primary renal tuberculosis. All of these must, of course, result from entrance of the bacilli through some portal, and in many cases the primary focus of disease is no doubt overlooked because of its insignificance rather than because none exists. There are, no doubt, cases in which the bacteria enter without producing a local lesion at the portal of entrance, but such instances must be comparatively rare. In a second sense, the lesion presented here is primary, viz., in its relation to the genito-urinary involvement. Its appearance and size alone would make it seem probable that the infection, if any connection existed between the various lesions of the genito-urinary tract, was descending, rather than ascending, and the position of the nodule in the kidney conclusively showed that the infection was not of the ascending type. *March* 12, 1896.

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3. *Hypertrophy of prostate gland in a man of thirty years, with suppurative cystitis and pyelonephritis.*

Exhibited by Alonzo E. Taylor, M.D.

These specimens were taken from a young man of thirty years who died of uremia after an illness of one year, in which prostatic symptoms were marked. The gland, as is here seen, is tremendously

enlarged, though symmetrically. I have made sections to determine whether or not the overgrowth was due to a neoplasm; it is, however, a simple hypertrophy of the glandular, muscular, and fibrous elements. There was no history of venereal disease, and the urethra presented no signs of stricture. *May* 14, 1896.

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4. *A case of diphtheria of the uterus.*

Exhibited by B. Franklin Stahl, M.D.

Mrs. K., aged twenty-two years; multipara. Delivered rapidly, after a brief labor, before my arrival. Perineum lacerated, and repaired at once. Temperature at once assumed a febrile type, and on the third day, notwithstanding intra-uterine douches and antipyretic measures, it was 105°. The discharge was extremely offensive and watery, with white and yellow membranous specks in it. The uterus was large, boggy, and tender on pressure. The cervix was covered with a yellow membranous deposit. Cultures from this region showed diphtheria-bacilli. There were no throat-symptoms. The pulse and respirations were proportionate to the fever. With slight remissions, the fever continued until the eleventh day, when it reached 107°, and she died. I was unable to discover the source of infection. I had no diphtheria-patients at that time, nor were there any cases in the neighborhood. *May* 28, 1896.

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5. *Anemic and hemorrhagic infarct of the kidney and spleen.*

Exhibited by J. Dutton Steele, M.D.

The specimens in this case were removed from a female, aged forty-two years, a native of Ireland. There are bright, white areas, irregular in shape, slightly raised above the surface, scattered irregularly over the cortex of the left kidney. It extends into the kidney-substance to different depths. The tissue is pale-yellow and hard. These areas have replaced almost all of the secretory substance at the lower half of the organ. They show no evidence of degeneration. The right kidney shows a single area of the formation above described at the lower portion of it. The cortex—not affected—is extremely anemic and somewhat fatty. The pyramids are congested.

Spleen. There is considerable old peritonitis. There is a whitish-yellow mass in the lower half of the organ, similar to that described in the kidneys. This is 7 c.m. long and 2 c.m. wide at its widest portion. It is closely incorporated in the tissues of the organ, and cannot be shelled out.

Microscopic examination of the formations in the kidney revealed fragmentation of the nuclei and swelling and obliteration of the cell-outline. In some places the nuclei have entirely disappeared and the cells are vacuolated. There is some round-cell infiltration around the edges of the diseased tissue. The formation of the spleen is much the same picture, except that there has been much hemorrhage and escape of red blood-cells into the affected area in addition to the cellular changes already described.

No reasons for the extensive infarction could be discovered. There was tuberculosis of the right lung, and a large cavity in the lower posterior portion. The liver is fatty, and there is some fibroid degeneration of the myocardium. *June* 25, 1896.

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6. *Malignant chronic endometritis.*

Read by invitation by J. M. Van Cott, Jr., M.D., of Brooklyn.

It is open to debate whether or not the title selected for this brief paper be correctly applied, but its choice has been governed by the constant relationship between the two conditions of inflammation and hyperplasia of the endometrium. Olshausen[1] has described the condition to be discussed under the title "Endometritis hypertrophica s. fungosa," which is similar to what Abel[2] describes, and is characterized by profuse proliferation of gland-structure, together with multiplication of capillaries in the endometrium.

Amann[3] describes it as follows: "The characteristics of these benign hyperplasiæ of the gland-elements are the facts of their superficial proliferation and the limitation of the growth to the tissues of the endometrium."

The same author further states that "aside from these benign alterations the surface proliferation of glands can, in rare instances, exhibit

[1] Olshausen, cit. Karl Abel, "Die mik. Technik u. Diagnostik in der gyn. Praxis." Berlin, 1895.

[2] Abel. Vide supra.

[3] Amann, Josef A., Jr. Lehrbuch der mikr.-gynekolog. Diagnostik. Munich, 1896.

malignant disease, *i. e.*, through transformation into adenoma, whereby one gland lies under another, with but little interstitial tissue, the progress of the growth being outward into the musculature."

Orth[1] has practically described the same thing when he says: "The finding of gland-tubes lying closely together, particularly when they lack the normal boundary of a tunica propria and longitudinally-disposed muscle-fibers, is sufficient for the diagnosis of a malignant neoplasm, which indicates hysterectomy."

Julius Schwalbe[2] has given a capital description of the lesion under title of "Malignant adenoma," as follows: A particular form of malignant new formation is the malignant adenoma—the gland-carcinoma of the corpus mucosum. It consists in excessive development of the glands of the uterine mucosa, which leads to complete disappearance of the interstitial tissue of the endometrium and to gland formation at the expense of the muscular tissue of the uterus.

"Even though metastases never occur in this form of malignant growth, its viciousness is evidenced, first, by the fact of repeated and unfailing recurrence after removal of the degenerate mucosa with a sharp curette; second, that this growth induces progressive destruction of the uterine parenchyma (see Figure); and third, that clinically, through persistent hypersecretion (brought about by gland-hyperplasia), and through extreme metrorrhagia, in the end cachexia and death supervene." "Typical carcinoma may also develop in the new-formed glands."

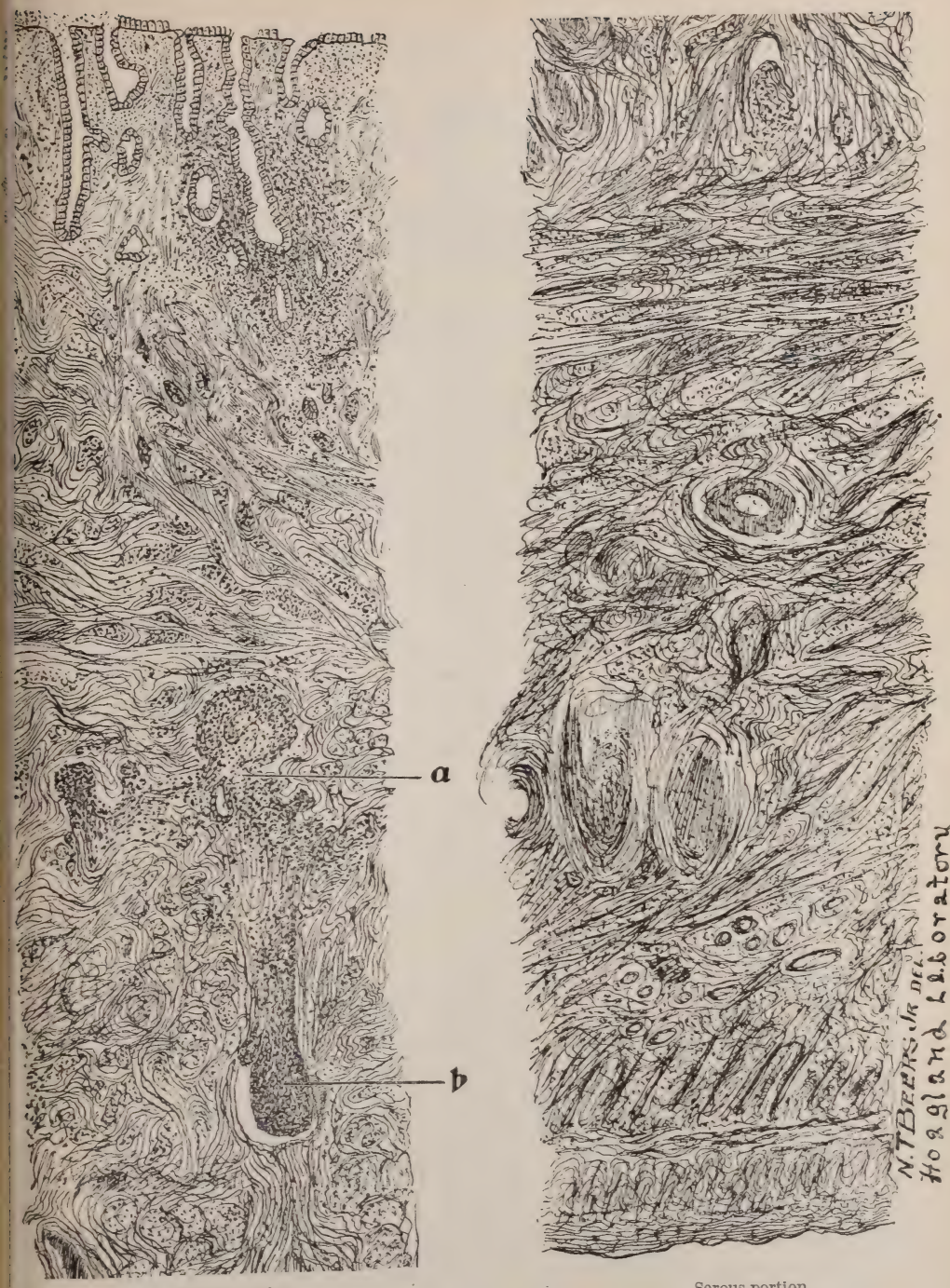
"The diagnosis is constituted by microscopic examination of curetted masses."

Schwalbe further says: "When, in the further course of the case, its recurrent nature is shown, *i. e.*, that a malignant adenoma is at hand, hysterectomy is indicated." A good deal is being said and written upon this subject at present; and the gynecologist is expecting more and more from the pathologist in point of differential diagnosis in all conditions involving hyperplasia of tissue in the female genito-urinary tract.

The average surgeon is not aware of the difficulties to be overcome by a microscopist in the successful examination of material dispensed to him by the surgeon's hands; for the reason, that he is too busy to become proficient in laboratory-methods. Every one who has had much to do with microscopic diagnosis has discovered this. But, assuming that material be furnished for examination in proper preservation, and from the right location, I would like to ask the question of those here

[1] Orth, Johannes. Allg. Path., 7th edit. p. 483.

[2] Schwalbe, Jul., Grundriss der spec. path. Anat. p. 524. Berlin, 1892.



Section through corpus uteri of Dr. Jenett's case, including all the structures. The endometrium is in hyperplasia. At *a* and *b* are seen portions of characteristic tissue of the endometrium which are seen to lie deep down in the muscularis, and near large bloodvessels.

whether it be possible, in their judgment, to find this condition with any degree of certainty *intra vitam* by means of microscopic analysis of the products of the curettage?

I would put the question in this way:

1. Can the diagnosis be made microscopically?

2. If so, can it be made before widespread adenomatous change has occurred?

3. If the microscope alone is incompetent, is there any combined method by means of which reliable results may be looked for?

In spite of the justice of the view, that to remove a uterus unnecessarily is a grave error, it has always seemed to me that to fail to remove it, the necessity being at hand, would be an even graver error.

Statistics and experience are teaching us constantly that conditions supposedly insignificant will some day assume grave proportions, and nowhere more than in the very class of cases under consideration. My own view of the matter after six years of observation is, that the microscope alone is not competent to determine the diagnosis of this form of malignancy *in utero*. While a great deal may be determined regarding the given case in point of the hyperplasia, the rapidity of cell-proliferation and the presence or absence of adenoma, it may be impossible to say whether or not the process is a superficial one, or already deep-seated, involving the intermuscular septa of the metrium.

Certainly the presence of mitosis, while it proves active proliferation of cells, will not show in curettings the location of the hyperplasia, nor will the general facts concerning the cells in their relation to each other do it. At best there can only be a strong suspicion of the presence of a condition which may prove fatal long before the morphologic changes are so clear as to leave no room for discussion or doubt as to their nature.

My own observation has extended over about a half-dozen cases in as many years of pathologic work in three hospitals in Brooklyn; and in one only of the six was I able to complete the investigation by examination of the enucleated organ. This statement is made for the purpose of emphasizing my belief in the comparative rarity of the cases.

With your indulgence a brief history of this case will be given:

The patient was aged about fifty, short, and very stout, with flabby tissues, and rather anemic. Her uterine history was that of profuse metrorrhagia extending over a period of five years, and also of increasing amount and duration, and lessening intervals. Repeated curettage was followed by recurrence of metrorrhagia and profuse mucoserous discharge *ex utero*. Microscopic examinations of the curettings revealed

an apparent simple gland-hyperplasia; and report was based upon the findings, with the request that, if curettement were repeated, more of the material be submitted for examination. In the course of a few months more curettings were sent, in a very fresh condition; and, after careful hardening, were sectioned, stained, and again examined microscopically, with the same result, *i. e.*, simple gland-hyperplasia.

In the report upon this second curettage I stated it as my belief that this was a case of malignant hyperplastic endometritis, advised another examination of material in case of necessity for a third operation; and suggested that if the findings on examination of the third specimen remained unchanged the case should be regarded as a good one for hysterectomy.

Not to absorb too much of your time—the case was operated upon, and I am able to show you sections through the wall of the uterus and to describe its condition, as follows:

The gross examination of this organ revealed it to be decidedly enlarged and somewhat globular in shape, with a relatively short cervix. The perimetrium was smooth, glistening, and everywhere hyperemic. Pressure conveyed the sense of softness and some elasticity. On gross section the cut surface was slightly rough, and appeared everywhere hyperemic, especially in and near the endometrium, which was considerably engorged with blood and hemorrhagic in spots.

Total thickness of uterine wall from surface of endometrium to surface of perimetrium a full inch and three eighths. The endometrium was soft and velvety in feel, and apparently one-fourth of an inch thick.

For microscopic purposes thin portions of tissue were cut with a large, sharp knife about one-eighth inch thick and one-half inch wide, stretching through the peri- and endometria, at a point just below the cornua. These were at once immersed on flat blotting paper into 95 per cent. alcohol; and, after hardening, embedded in celloidin, and sectioned on a Thoma microtome, and stained with hematoxylin and eosin, and mounted in xylol-balsam. There was marked shrinkage of the tissue in the alcohol, especially of the endometrium.

The microscopic findings are quite in keeping with those of the curettings. There is no traceable evidence of either carcinoma, sarcoma, or adenoma; but everywhere is evidence of gland-hyperplasia, not extreme, but showing the distinct tendency to outward growth into the intermuscular spaces of the metrium. An interesting occurrence is the growth of the matrix of the endometrium far into the metrium. This is not described in these conditions in the various reports of them that I have been able to examine. I believe that I have seen areas of this portion of the endometrium growing very close to the perimetrium, and am

now engaged in an effort to find a differential stain that will help to prove the correctness of this finding.

The general impression conveyed by the microscopic findings is that of a progressive *outward* hyperplasia of the corporeal endometrium. This hyperplastic tissue is evidently displacing and replacing the muscular tissue of the metrium, and its proximity to the bloodvessels must be regarded as a possible source of the metrorrhagia.

When the honor of an invitation to read a paper before this Society was extended to me, I made up my mind to bring this matter up before it for the benefit to be gained from the experience of its members concerning it.

You will all doubtless concur in the proposition that, notwithstanding the difficulties and often impossibilities in the way of forming any kind of reliable conclusions from the examination of uterine tissue removed for purposes of diagnosis, it is nevertheless the duty of a pathologist to "stand by his guns" in such cases, in the hope of discovering and possibly routing the enemy.

After mature deliberation my personal belief is that the diagnosis of endometritis chronica malignans cannot be positively made in the present state of our knowledge by means of the microscope alone; but that with the combined microscopic examination of the curettings and a good clinical history it can be determined.

Discussion.—Dr. McFarland in discussing Dr. Van Cott's paper said he had had similar experience in that the number of cases is very limited. The condition must occur rather rarely. Unfortunately, too, we could never study what the process would have amounted to if the organ had not been removed. Another very important question was whether this pathologic process was of the nature of a neoplasm or of an inflammation with hyperplasia. If this hyperplasia could go on and extend into the endometric tissue, without penetrating beyond, he would regard it only as an inflammation. He did not think our present knowledge was sufficient to say just what the process was. In a majority of cases the microscopic appearance was like that of a neoplasm. He was accustomed to teach that, when the epithelial cells were regularly arranged on the membrana propria, preserving their normal shape and with their nuclei near the basement-membrane, the process was benign. On the other hand, if the cells were crowded and did not have proper room, so that some were detached and were found lying in the center, and the nuclei took a central position in the cells, then the cells were growing too rapidly, and probably would soon escape into the surrounding tissue. In

such cases he always advised extirpation of the uterus, but, of course, he could not say what would be the result if such an organ were allowed to remain. *October* 27, 1896.

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7. *Primary interstitial nephritis in a young man, without arterial change. Peripheral neuritis.*

Exhibited by J. P. Arnold, M.D.

The patient was a young man, aged twenty-two years, admitted to the Presbyterian Hospital, August 4, 1896, complaining of inability to walk, because of loss of power in the left leg for the past week. He also complained of weakness in the left arm, headache, nausea, and vomiting, and general malaise. When the man was admitted he was in a stuporous condition; speech was slow and thick, almost unintelligible. The pulse was increased in tension, but the artery felt normal. There was almost complete loss of power in the arms and in the left leg. There was some evident wasting of the muscles and tenderness over the nerve-trunks. The urine was light in color; specific gravity, 1009. The amount varied from 53 to 119 ounces daily. At first, albumin was not found, but later was present to the extent of 0.1 per cent. No casts were seen at any time. The stuporous condition increased to coma, and the man died two weeks after admission.

Autopsy. The postmortem examination was made four hours after death. The body was quite markedly emaciated; no edema was present. Heart weighed 300 grams. There was no endocarditis, except a slight thickening of the mitral leaflets. The mitral orifice was rather small, admitting only two fingers. The openings of the coronary arteries were situated a considerable distance above the aortic valves, and were not covered when the leaflets were laid back against the aortic wall.

Kidneys. The right weighed 80 grams, the left 75 grams. They were very firm in consistence; capsules stripped with difficulty. They both presented on their convex surfaces a peculiar enlargement which made them almost pear-shaped. Section showed almost entire absence of renal structure. In the right, one or two pyramids could be distinguished; in the left, none. The surface of the section was almost gristly. The color was rather paler than normal. In the left, toward the periphery, were numerous small, yellowish areas which were some-

what denser than other portions. In the right there was a small cyst with clear contents.

A peculiarity about the blood was noticed which seemed worthy of mention. When the hands, covered with blood, were brought in contact with water, the blood changed to a brownish color, as though there had been some alteration in the blood-pigment. There was no apparent arterial disease, though the arteries were not examined microscopically.

There was nothing in the case to account for the condition of the kidneys. He used no alcohol, and denied all venereal history. He stated that his health had always been good until the time of the present illness. I do not know what to make of the condition, unless it was congenital.

Contracted kidney in the young is a very rare condition. When it does occur, it is usually accompanied by very marked arterial degeneration, and hypertrophy of the heart. In the present case, the heart was normal in size, and there were no arterial changes to be found.

The location of the coronary arteries is of very great interest. It seems to me that there can be no doubt that, in this case, at least, they were filled during the systole of the heart. *December* 10, 1896.

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8. *Papilloma of the bladder, causing death by hemorrhage.*

Exhibited by David Riesman, M.D.

The history of this case, which was kindly furnished me by Dr. McCarthy, is as follows:

H. L., aged forty-six years; colored; an American by birth; had the usual diseases of childhood; gonorrhea at eighteen; some venereal sores at twenty-two, without secondary symptoms. For three years has had leukoderma of hands, face, and neck. Alcoholism is denied.

Since September, 1896, he has had pain in the back and in the lower part of the abdomen, and also noticed the presence of blood in the urine. Occasionally the urethra would be blocked by clots, especially toward the end of micturition. Lately he has been passing more blood, and had to give up work on account of increasing debility. There is no pain during or after micturition; the quantity of urine is normal.

At the time of admission he was anemic and apathetic; the lungs were normal; at the end of the sternum a depression (congenital) was seen. The apex-beat was in the normal position; a presystolic thrill

could be felt at the depression noted; the impulse was weak. Over the mitral area a systolic murmur was heard and could be traced to the axilla; there was also a diastolic murmur at the same area, but less distinct. Over the depression, at the end of the sternum, a presystolic murmur was heard. A loud systolic murmur was also audible in the third interspace to the left of the sternum; it was not transmitted. There was no venous pulsation, venous stasis, or dyspnea.

The abdomen was normal.

The urine had a specific gravity of 1024; contained abundant red blood-cells, but no casts. On standing, a sediment of blood formed on the bottom of the glass.

On the day following his admission the patient had a sinking spell and almost fainted; free stimulation restored him somewhat. At 12.30 P.M. he had another attack, characterized by restlessness, delirium, and hunger, and extreme pallor; the pulse was weak and running, and soon became imperceptible. Death took place in half an hour from the beginning of this attack. Percussion at this time revealed no dulness above the pubes.

The autopsy was made twenty-four hours after death.

Pathologic diagnosis. Anemia of all the organs; fatty degeneration of heart; chronic parenchymatous nephritis; papilloma of bladder; hemorrhage into cavity of bladder; hypertrophy of prostate; plastic pleurisy of left side; tuberculosis of lymphatic glands in superior and posterior mediastinum; atheroma of aorta; papillomatous tumor of stomach; accessory spleens and pancreas.

Autopsy-protocol. Body of a well-developed negro; rigor mortis well marked; small pigmented scars on right tibia; old scar on right side of chin. Peritoneum is quite dry; stomach dilated; appendix lies parallel with and somewhat in front and to the left of the ascending colon, making an upward bend on itself from its origin; it has a mesentery of its own. There are adhesions between the ascending and transverse colon and under surface of gall-bladder.

Left lung adherent the whole distance from above downward, from anterior axillary line back. The right pleural cavity contains 90 c.c. of a straw-colored fluid; no adhesions on that side.

In the anterior mediastinum, just above the pericardium and behind the left brachiocephalic vein, there is a large lobulated gland, on section presenting a mottled appearance—a greenish substance traversed by yellowish-white bands.

External layer of pericardium is infiltrated with fat at base of heart. There is a slight excess of fluid in the sac. Bands of adhesion pass

between the layers of the pericardium reflected from the aorta to the parietal layer.

The right auricle is enormously distended, containing fluid blood and chicken-fat clots. Tricuspid orifice admits four fingers; left auricle contains liquid blood, currant-jelly and chicken-fat clots; mitral orifice admits three fingers.

Aortic valve leaks slightly when hydrostatic test is employed; the intima of the aorta shows small patches of atheroma. Pulmonic valve normal; right ventricle measures 5 mm. in thickness, is soft and flabby; left ventricle 22 mm.; its muscular substance is very pliable and yellowish in color. Weight of heart, 410 grams.

Left lung adherent to diaphragm; crepitant throughout; emphysematous at margins and apex. Interlobar adhesions. Bronchial glands much enlarged and very black. Lung pale; mucous membrane of bronchi pallid. Weight, 410 grams.

Right lung. Very edematous, more so than left; some hypostatic congestion at base; bronchial glands enlarged and anthracotic; three of the upper ones present tubercles. Weight of lung, 560 grams.

Spleen. Capsule wrinkled; pulp pale; in upper part a small fibrous nodule; there are two supernumerary spleens just over the apex of the left kidney. Weight, 130 grams.

Left suprarenal quite firm.

Left kidney. Rather small; capsule strips with difficulty, but does not tear cortex; at junction of upper and middle thirds an old stellate scar. On section, advanced fatty degeneration, cortex yellowish-white and slightly diminished in thickness; surface smooth; substance soft.

Right suprarenal slightly yellowish.

Right kidney larger than left; capsule strips easily; on section practically like the other.

Ureters normal.

Bladder distended; extends three-fingers' breadth above pubes. On opening find considerable quantity of bloody urine. Chief contents a large dark-red blood-clot, weighing 200 grams. On removing this there is found on the left wall of the bladder, 22 mm. from mouth of left ureter, a flat papillomatous tumor, 27 mm. long; greatest breadth 15 mm.; thickness about 3 mm.; pedicle quite firm, but mass of papilloma soft, and convex edge shreddy. The clot was directly in contact tact with the polyp, the latter unquestionably being the source of blood.

At vesical mouth of urethra there is a sessile mass the size of a cherry, partly occluding the urethra; it is evidently an enlarged middle lobe of the prostate. Mucous membrane of bladder congested and somewhat thickened. Lateral lobes of prostate much enlarged.

Stomach dilated, pale; 3 cm. from pylorus a sessile papillomatous mass size of a small grape; it is freely movable.

Pancreas. Small accessory pancreas, lying above and to left of main pancreas; has a duct of its own three inches long; pancreas very pale.

Liver. 1430 grams; 23 cm. long; right lobe 17 cm. wide, left 15 cm.; thickness 9 cm. Edges sharp; substance firm and pale. Surface marked by small whitish areas; such areas also found in substance of organ. Two small black areas on surface, possibly angiomas.

Intestines pale; show nothing abnormal.

Brain. Calvaria unusually thick; brain pale and firm; dura not adherent.

REMARKS. The interesting feature of this case is the tumor of the bladder, which, although quite small, was the indirect cause of the death of the patient. Tumors of the bladder are on the whole quite rare. Gurlt,[1] among 16,637 tumors of all kinds, found 66 that were vesical—a proportion of 0.39 per cent. Men are affected more than twice as often as women. The tumors of the bladder have been variously classified; the classification adopted by Thompson[2] is as follows: myoma, fibroma, myxoma, sarcoma, papilloma, carcinoma. Of these the form of papilloma known as the villous papilloma is the most common. Rokitansky applied the term villous cancer to these growths; but Virchow[3] proved that such a designation was erroneous. In addition to the tumors named, there is on record a case of papillary adenoma (Kaltenbach), and dermoid cysts have also been described.

From the clinical standpoint tumors of the bladder, even when histologically benign, are of serious import, leading to grave if not fatal consequences, either by hemorrhage or by obstruction of the urethra and surgical kidney.

As elsewhere in the body, the tumors of the bladder are found generally about the orifices, and in the case here reported the growth sprang from a point near the mouth of the left ureter.

The hemorrhages in vesical growths are often entirely painless, and come when the patient is perfectly quiet, at times during sleep. In some cases there is pain, especially when the urethra is plugged by clots. The urine shows the character of vesical hematuria, which I need not describe. At times, particularly in the case of villous papillomas, portions of the tumor are passed with the urine.

As to the nature of the present growth, I am unable to make any positive statement, as, in order to show it in its entirety, I have not made any microscopic study. It may be a vascular papillomatous fibroma.

The amount of blood in the bladder of this case would not have been sufficient to cause death had the patient not been rendered very anemic

by the previous loss of blood, extending over two months. Nearly all writers refer to the possibility of death from anemia and exhaustion, but as far as my search of the literature has gone, instances like this, in which death was sudden, are very rare.

Dr. Pepper,[4] in 1871, reported a case to this Society, in some respects almost paralleling the present one. The patient had had an intermittent, apparently causeless, hematuria for some time; on October 29th the final bleeding began, and continued until November 8th, ten days, when the patient died. At the autopsy, barring general anemia, nothing abnormal was found anywhere, except in the bladder; this viscus was distended with blood-clots; the walls were everywhere healthy, except at one spot, about one and one-half inches in diameter, near the fundus, at which a soft fungous growth, about the size of a half-walnut, was found. Its color was pinkish, and it was evidently highly vascular. The Committee on Morbid Growths declared it a benign papillary growth.

Wardell[5] reports a case of malignant disease of the bladder; the patient also dying of hemorrhage. At the autopsy the bladder was found to contain about five and one-half pints of blood; from a small cavity communicating with the main cavity of the organ two or three ounces more of blood were obtained.

Langhans[6] records the case of a youth, aged nineteen years, who had suffered from intermittent hematuria for ten years, coming on generally once a year in the spring. The final hemorrhage lasted only ten days, and at the autopsy cavernous tumors and varicose veins were found in the wall of the bladder. The bleeding had occurred from the cavernous tumors, which involved the mucosa and submucosa.

Sömmering[7] refers to two cases dying of hematuria, but does not give the cause of the latter.

The tumor of the stomach referred to in the notes is one of the nature of which no definite statement can be made without microscopic examination. It might be a myoma.

Of some interest, also, are the supernumerary spleens and the accessory pancreas, the so-called lesser pancreas.

Literary References.

1. Gurlt. Quoted by Ultzmann. Deutsche Chirurgie, xii. p. 310.
2. Thompson. Tumors of the Bladder, 1884, p. 66.
3. Virchow. Die krankhaften Geschwülste, Bd. iii.
4. Pepper. Philadelphia Medical Times, 1871, ii. p. 115.
5. Wardell. The Lancet, London, 1850, i. p. 423.
6. Langhans. Arch. f. path.-Anat., 1879, lxxv. p. 291.
7. Sömmering. Tödtliche Krankheiten der Harnblase und Harnröhre, Frankfurt a. M., 1822, pp. 156, 159.

January 14, 1897.

9. *Tuberculosis of kidneys.*

Exhibited by S. M. Hamill, M.D., through the courtesy of
John A. Johnston, M.D.,
Assistant Surgeon, National Soldiers' Home, Va.

The only clinical history obtainable in this case consists in the report that the patient died of starvation as the result of a refusal to take any nourishment. The urine had been repeatedly examined for tubercle-bacilli, with negative results. Oxalates, blood, and pus were always found.

The autopsy, made five hours after death, by Dr. Johnston, gave the following results:

Andrew F., white; aged sixty-four years. Body much emaciated. Postmortem lividity in the dependent portions of the body well marked. Rigidity was absent.

Thorax. Costal cartilages were ossified.

There was a small patch of pleural thickening over the lateral surface of the upper portion of the lower lobe of the left lung. On section, the lung exuded a frothy, reddish-colored liquid. There was a healed tuberculous lesion at the apex of the right lung.

The heart seemed under the normal size; the vessels were engorged, and the heart was covered with fat. The pericardium contained about one dram of straw-colored serum. The right auricle contained a currant-jelly clot. The cavity was normal in size. The tricuspid orifice admitted the tips of two fingers. The cavity of the right ventricle was about normal in size and contained a currant-jelly clot.

The walls measured $\frac{3}{8}$ of an inch in thickness. The muscle was pale and of an oily feel. The tricuspid leaflets were thickened, and the chordæ tendineæ were contracted. The left auricle was smaller than normal, and contained fluid blood. The mitral orifice admitted the tips of two fingers. The cavity of the left ventricle was contracted and its walls thickened, measuring $1\frac{1}{4}$ inches in thickness, and showed signs of fatty change.

The aortic and pulmonary valves were normal. The coronary arteries were normal. There were a few small patches of atheroma in the aorta.

The liver was pale on the surface. On section it imparted a gritty sensation to the touch. The gall-bladder contained two ounces of bile, and the gall-duct was patulous.

The spleen was of normal size; its capsule was thickened and adherent.

The left kidney was difficult to detach. It was about normal in size and outline, and the capsule was very adherent. Section of the kidney revealed a number of cysts, which in the fresh state contained greenish, curdy pus. The largest cavity contained a small black stone about the size of a pea. The left ureter was somewhat thickened. The thin, smooth cyst-walls in this kidney, the absence of tuberculous deposits between the cysts, and the presence of a small calculus in one of the cysts would lead one to look upon this rather as a surgical than a tuberculous kidney.

The right kidney formed quite a tumor-mass in the right abdomen. The greatly thickened perinephric tissue was tightly adherent to the kidney and to all the surrounding structures. Its relation to the liver was so intimate that it had to be dissected away, and, in so doing, a large abscess-cavity was discovered which communicated with the pelvis of the kidneys. The kidney was greatly increased in size, and was embedded in the augmented perinephric tissue. This fibro-adipose capsule was so adherent to the true kidney-capsule that it was impossible to differentiate them. The whole mass was tightly adherent to the kidney-structure and tore the latter in stripping.

Section of the kidney showed extensive tuberculous infiltration, with almost complete destruction of the kidney-tissue. The tubercles varied in size and degree of degeneration. Some of them had softened completely and formed abscesses, which in the fresh state contained greenish, curdy pus.

The pelvis was somewhat dilated; its mucous membrane thickened and rough. Its communication with the abscess-cavity, lying between the kidney and liver, caused it to be filled with greenish, purulent matter containing some caseous material.

The ureter was thickened throughout, irregular in outline, and its lumen here and there almost obstructed by masses having the appearance of blood-clots. The mucous membrane of the bladder in the region of the trigone was thickened. Frozen sections from this region were stained by Dr. Johnston for tubercle-bacilli, with negative result. The bladder elsewhere was normal.

The mesenteric glands were somewhat enlarged, but not cheesy. The bronchial glands were not enlarged. There was no involvement of the genital organs.

The area of healed tuberculosis observed in the apex of the right lung is of interest in its relation to the mode of infection. It suggests that the kidney-condition may be entirely independent of this lesion, and a new infection from some other source. The involvement of the

mesenteric glands would lead one to consider the possibility of an independent infection through the gastro-intestinal tract.

Whatever may be the origin of the infection, a study of the genito-urinary tract shows the kidney primarily affected and the bladder involved by descending infection. This finding is in keeping with the conclusions reached as the result of an exhaustive study of the subject of tuberculosis of the kidney made by me and published from the Pepper Laboratory of Clinical Medicine in the early part of last year.

Discussion.—Dr. Steele asked what Dr. Hamill considered the source of infection. Dr. Hamill, replying, thought it was doubtful whether the infection was from the old healed lesion in the lung or from the apparently more recent involvement of the mesenteric glands.

January 28, 1897.

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10. *A tubal gestation sac containing fetus.*

Exhibited by Henry L. Williams, M.D.

The history of the patient from whom this specimen is presented is as follows:

E. K., aged twenty-three years; born in Germany; came to this country a little more than three years ago; was married in June, 1894 (two and a half years ago). First child born in March, 1895. Up to time of birth of child was perfectly well; never sick a day. Sicknesses regular every month; lasted two or three days, attended with no pain; never had to go to bed on account of them. After child was born patient got up in ten days and began to go about. She began to have pain in left ovarian region immediately on getting up. Her sickness did not return for six months. She nursed her baby until July, 1896 (sixteen months). Her bowels have always been regular and in good order. She has never had any leukorrhea or vaginal discharge.

Since the birth of first child, March, 1895, her sicknesses have lasted from four to seven days, with menorrhagia. They have usually been attended with so much pain that she has been obliged to lie down most of the second day. The pain sometimes lasted a week. This pain was only in the left side.

On August 16, 1896, she aborted a nine-weeks-old fetus. After this she had no menstrual flow for three weeks. She then had a constant

profuse flow for two weeks. This stopped about the third week in September.

In the last week in October another regular sickness began, but stopped suddenly, after only two days' flow. On November 6 she had a slight "show," which lasted only a part of one day.

From this time until November 28 there were no manifestations. On this morning, at about 9.30, she began to scrub the floor on hands and knees, and had been working only a few moments when she was taken with such sudden violent pain in the left ovarian region that she fainted. She revived in about ten minutes and took a little whiskey and ginger. Severe pain lasted until noon. She then felt much better, and finished scrubbing the floor. On coming to she had felt sick at her stomach, and vomited.

This was on Saturday. On Sunday and Monday she had no symptoms. About noon on Tuesday, while at work, she was taken by another severe pain in the left ovarian region, similar to the first, but she did not faint. This lasted all the afternoon. A doctor was sent for and medicine given, which relieved the pain. There was some pain on the following day, but much less severe.

There were scarcely any symptoms after this, aside from a constant slight pain in left inguinal region, until Thursday, December 10, when there was a profuse hemorrhage from the vagina. The doctor was summoned and obliged to insert a tampon to stop the flow. On Friday the flow continued, and a fleshy mass was passed, half the size of the thumb. On Saturday, the 12th, the flow suddenly stopped, and there was some pain. Patient was told to remain in bed, but was obliged to get up and move about the room to prepare the meals.

On Sunday and Monday there were pains, but not so severe. On Tuesday patient felt pretty well, and was given permission to go about. On Wednesday, December 16, she felt well in the morning. In the afternoon, about 3 o'clock, she lay down on the bed beside her seventeen-months-old baby to rest. The child was playful, and in sport suddenly jumped on its mother's abdomen.

The woman had immediate intense pain, and felt as if something inside had "cracked." She arose and sat in a chair, then walked the floor with most intense pain. She then again sat in the chair, but soon became so stiff that she could not rise. She described her sensation as if being "broken in two in the middle." Her brother-in-law happened in, and carried her to bed in his arms. The pain all this time was excruciating. A neighboring physician was called in in the emergency and prescribed whiskey ʒj. every ten minutes. The regular attending

physician arrived at about 5.30 P.M. At this time the whole abdomen was sensitive to the touch, but the point over the left ovary exquisitely painful to the slightest contact.

Neither physician made a vaginal examination (and none had been made previous to this time), but at this time a diagnosis was made of internal bleeding. Ergot was given, and a teaspoonful of whiskey and water in equal parts every fifteen minutes all night. Ergot was also given every hour and a half during the night.

At 1 A.M. the patient fainted and remained unconscious for an hour, so that the husband thought that she was dead, and cried out. This aroused the woman, and stimulants were continued. The night was passed in intense pain, without sleep. The bowels moved once during this time.

During the following day whiskey was given every half-hour, and arrangements made for operation. The patient was carried to the Howard Hospital and operated upon by Dr. B. C. Hirst at 5.30 P.M. The operation was performed under partial anesthesia with the utmost celerity, and occupied but twelve minutes. On opening the peritoneum the abdominal cavity was found to contain large quantities of partially clotted blood. A small gestation-sac, no larger than a walnut, was found in the left tube, containing an embryo probably less than forty days old. The tube and ovary were removed. On the posterior upper surface of the tube is seen a small fissure, about an eighth of an inch in depth and a quarter of an inch in length. From this small area the bleeding had occurred. The sac was not torn through.

A few clinical features connected with the operation are also of interest. When placed upon the table the patient's pulse was about 180, feeble, and fluttering. When placed in bed after the operation the pulse was 110 and fairly strong. This is attributed to the large quantity of hot water left in the abdominal cavity. The pulse, however, soon again failed, requiring active stimulation and repeated use of usual salt solution per rectum. The woman, however, survived and made a good recovery. *February* 11, 1897.

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11. *A contribution to the pathology of albumosuria.*

Presented by A. E. TAYLOR, M.D.

In this paper I use the word albumosuria to signify what has, up to within a few years, been known as peptonuria. The word peptone was originally used to designate all forms of proteid which could not be

precipitated by potassium ferrocyanid in the presence of acetic acid; the studies of Kühne and his students have since thrown much light upon the lower proteids, and it has been shown that compounds exist which resist even more precipitant salts than potassium ferrocyanid, and to these the name of peptone has been transferred, while the older peptones have been shown to be one or the other of the albumoses, usually deutero albumoses. In this connection I may remark that the peptone of Witte and the later preparation of Adamkiewicz, at least in the samples I have analyzed, do not contain any of the peptone Kühne, but are composed for the most part of deutero-albumoses, although a little of each will pass through a neutral saturation with ammonium sulphate.

There are a vast number of studies in literature upon peptonuria and its clinical significance. In many of these the methods employed were such as to make the result of no definite value, while in the majority of all no attempts were made to differentiate albumoses from peptone. Within recent years, however, such able chemists as Kühne, Hüppert, Mörner, Salkowski, and Neumeister, together with Senator, Hubel and Matthes and Stadelmann have shown, in the first place, that under the use of stricter and more elective methods albumoses are much rarer than even peptone previously was, and that in all cases it is necessary to confirm the biuret reaction, or to put the urine through a long process before its application, in order to render it trustworthy. They, furthermore, were in no case able to find Kühne's peptone in the urine, and I know of no such observation up to to-day. Attempting to get at the bottom of pyogenic peptonuria, so-called, chemists set to studying the affected tissues and pus, most often with negative result; such studies have, however, been few, and this it was which suggested the studies, a partial report of which I wish to present to-night. The material of which I report has consisted of pus and diseased tissue of twelve cases, and of the urine of four cases of peritonitis. The methods I have employed are those worked out by Kühne and Neumeister, and of them a brief description is necessary.

The pus or tissue, which was always fresh, was extracted with warm distilled water, and the coagulable proteids removed by heat. This is, perhaps, the most trying part of the entire method, completely freeing the specimens of all coagulable proteid. The mixture was first gently heated to 90° C., then very dilute acetic acid carefully added, and then boiled and filtered. Then enough acetic acid was added certainly to convert the albumin into the acid reaction, after which, with the temperature maintained at 95° C., the mixture was carefully titrated with very

weak alkaline solution until another precipitation appeared, following which the mixture was made alkaline, and then carefully titrated back with weak acetic acid to see whether any further precipitate would appear. By working in this way I was able to remove the coagulable proteids in nearly all cases, so that Heller's test and a weak potassium ferrocyanid solution gave no reactions.

The urine was freed of albumin in the same way, following which both were treated alike. After cooling the solution is saturated with neutral ammonium sulphate and filtered, the filtrate being reserved for search for peptone. Any precipitate upon the filter paper is then dissolved in distilled water and dialyzed until the sulphate reaction has disappeared. In case a precipitate occurs during dialyzation it will be hetero-albumose, at least that has been the one general statement; but at the time in my series when such a precipitate occurred it refused all proteid reactions, and I have no idea what it was. Acetic acid is then carefully added to the salt-free solution, and by it nucleo-albumin, which in all my cases of pus was present, is thrown down and removed by filtration. A small portion of the clear filtrate is then mixed with an equal volume of a saturated aqueous solution of sodium chlorid, and acetic acid carefully added, after which it is boiled. If there was a precipitate or a cloudiness which cleared up in part on boiling, that serves as a general indication of the presence of albumoses. The remainder of the filtrate is then neutralized and saturated with rock-salt; if it remains clear there are no primary albumoses, if it clouds such are present, and should be filtered off, redissolved, and should respond to proteid reactions. To the solution, if no precipitate of primary albumoses forms, or to the filtrate after removal of them, there is then to be added so long as a precipitate forms a solution of strong acetic acid saturated with sodium chlorid. In case primary albumoses were not present any precipitate consists of deutero-albumoses; otherwise it will also contain some primary albumoses which are not fully precipitated by the sodium-chlorid process. This precipitate should be treated with the proteid reactions. In the filtrate proteid may still be present, since certain deutero-albumoses cannot be precipitated by rock-salt and acetic acid. It is to be freed of salt and acid by dialysis and any proteid present precipitated by alcohol or tannic acid.

The filtrate of the ammonium sulphate saturation is then to be studied for peptone. To a small portion an equal volume of distilled water is added, and drop by drop a fresh solution of tannic acid, avoiding an excess. If within three hours no precipitate forms the solution contains no proteid. If a precipitate forms it may be either low deutero-

albumoses or true peptone. To precipitate the deutero-albumoses the original filtrate is boiled, saturated with ammonium sulphate, made alkaline by ammonium hydrate and carbonate, allowed to cool and filter, whereby they are nearly always precipitated. If not, the ammonia is to be driven off by heat, the solution made acid by acetic acid and again boiled with an excess of ammonium sulphate, and with this all deutero-albumoses will be entirely precipitated. After this true peptone is to be tested for after removing the salts which hinder the reaction. I have never had occasion to go any further than this, as I have never seen a precipitate with tannic acid after the successive neutral, alkaline, and acid saturations with ammonium sulphate. If a proteid be present it should be precipitated by tannic acid, the sulphates removed with barium so carefully that the solution contains neither sulphate nor barium, the tannic acid removed with lead acetate, and the clear filtrate submitted to the biuret test with very strong alkali and very weak copper-solutions, and to Millon's reagent. Or it may be precipitated by alcohol, the sulphates removed as before, the alcohol evaporated, and again tested with biuret reaction or tannic acid, or dried in a vacuum with sulphuric acid.

As I have said, I have never found any peptone to use those methods upon. The entire method is obviously long and vexatious; it is, however, necessary, since it has been shown that there are unstudied proteid and other substances which respond to the shorter methods of Devito, Hofmeister, etc. And it must be further insisted upon that without the long series of isolating processes the biuret test is not trustworthy, and in the course of these studies I have several times, early in the process, seen faint biuret reactions, which were confirmed by my laboratory colleagues, when later there was shown to have been no albumoses or other known proteid present. For the material I am indebted to Drs. Stengel, Frazier, White, and Penrose.

Case I.—℥j. pus of septic origin, note of source lost; no reaction for any albumose.

Case II.—℥j. pus from septic abscess of axilla, a faint reaction of deutero albumose.

Case III.—℥j. septic abscess, no albumoses.

Case IV.—℥ss. tuberculous pus from neck, no albumoses.

Case V.—℥ss. pus from superficial abscess, entirely negative.

Case VI.—℥j. tuberculous pus from groin, albumoses present; reactions remained until the alkaline saturation with ammonium sulphate.

Case VII.—℥ss. pus from tuberculous abscess of neck, primary albumoses

Case VIII.—Solution of a fresh tuberculous kidney removed by operation; this contained both primary and deutero-albumoses in abundance.

Case IX.—℥j. pus of septic abscess, faint reaction of primary albumoses.

Case X.—℥x. of contents of a pancreatic cyst, which contained 7 per cent. of coagulable proteid, neither albumoses nor peptone, a fact which is striking when we consider the action of trypsin on albumin. The diagnosis of a pancreatic cyst was made entirely upon anatomic grounds at the operation, and no study of the contents was made to confirm it.

Case XI.—℥iv. pus from septic empyema, secondary to pneumonia; no albumoses.

Case XII.—℥viij. pus from kidney, eighteen hours after death, full of deutero-albumoses. This case, of course, cannot count, since the albumoses might have formed post mortem.

I have further studied the urine of four cases of septic peritonitis: one in a man, three in women, in one of whom it was secondary to a widespread necrosis of a pelvic neoplasm. In this last case deutero-albumoses were present; in none of the others.

To summarize: Out of eleven cases of fresh pus, albumoses were present in five cases. Of the cases seven were septic, and of these two had albumoses; four were tuberculous, and of these three contained albumoses. The negative results surprised me, but at the suggestion of Dr. Miller I, early in the studies, looked into the purity of my reagent, since mixtures of salts are known to precipitate inordinately, but no faults could be detected.

Albumosuria may be conceived as produced in three ways. Apart from possible proteid decomposition within the bladder, albumosuria presupposes albumosemia; but the converse need not be true, since there is some evidence that a slight albumosemia is tolerated by the kidneys without elimination.

The first way is the enterogenic albumosuria, in which the albumoses of digestion are absorbed unchanged, instead of being reconverted to coagulable proteids as normally. This has been held to occur in conditions attended with ulcerations in the gastro-intestinal mucosa. It can, however, never be extensive, since the major part of proteids is assimilated as syntonin, with only a moderate reduction to primary albumoses, and usually there is none below these. The albumosurias of phosphorus-poisoning, leukemia, and osteomalacia are by some held to belong to the enterogenic type, but it is rare in all of them.

The second way is by bacterial action, the infectious albumosuria.

That many bacteria can reduce albumin to albumoses is well known, particularly of the tubercle-bacillus. Whether bacteria produce albumoses by a direct action on the albumin as enzymes or whether they elaborate it as a part of their metabolic economy has not been determined. As stated, in the list of my cases it occurred in five, although I would not venture to assert too strongly that where it is absent in the pus it did not exist in the blood, since it may be possible that being so easily diffusible the albumoses may be carried away as soon as formed. Even when they are in the pus it is obvious that they remain there only a little while; there must be a constant outgoing into the circulation and a constant production, otherwise they would be reduced to true peptones.

The third possible method is by metabolic alterations apart from bacterial actions, a possibility first opened up by the study of idiopathic cystinuria. Recently the Jena school, Stintzig, Krehl, and Matthes, has attempted to show connections between fever *per se* and albumosuria. They contend, to eliminate the question of bacterial fevers, that the febrile reaction caused by injections of aseptic substances, such as silver nitrate and iodin, is always accompanied by albumosuria, both in animals and in men, in whom, for example, injections of iodin into a hydrocele occasion the phenomenon. They do not use the salt-methods, but precipitate the albumin-free urine with alcohol, and test the redissolved precipitate with the biuret reaction, and in a few cases also with the test described for primary albumoses, with entirely contradictory results, which they have not chemically explained. It is obvious that much work must yet be done along these lines. *February* 11, 1897.

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12. *Uterus bicornis unicollis.*

Presented by Joseph McFarland, M.D., and Wm. W. Babcock, Jr., M.D.

Clinical history. Miss Y., aged eighteen years, of slight build and apparent imperfect development; has never menstruated through the vagina or vicariously, although menstrual molimina, consisting of heavy bearing-down pains in the pelvis, associated with headache, have occurred each month. During the past year there has been a constant sharp pain in the pelvis, and one month ago she developed a marked leukorrhea—apparently the sign of a specific vaginitis.

Despite the decided sexual hypoplasia, the patient has been a prostitute, and she states that the sexual appetite is present.

Physical examination. The external genitalia appear normal. On examination before operation the vagina was found to be a cul-de-sac, about 3.5 cm. in depth.

After the abdominal section the patient apparently had an abscess discharge per vaginam, and another examination was made. At that time, beside the short, narrow vagina, an opening was discovered in the anterior vaginal wall, which admitted the finger and opened into a small cavity. Toward the upper part of this cavity a small opening was found, admitting the tip of the finger. It is possible that this last opening was the point of discharge of the abscess. No definite cervix could be made out, nor could the uterus or ovaries be mapped out per vaginam before the operation.

The lower opening, which was in the anterior vaginal wall, probably existed, but was overlooked before the operation.

Celiotomy was performed by Dr. Charles P. Noble, who found two small firm masses in the place of the uterus. These were united below to a fibrous cord supposed to represent the cervix. Each of these masses had attached to its outer part a Fallopian tube and a round ligament, demonstrating that they were uterine masses. Connected with the right side was a clear fluid parovarian cyst, the size of a plum.

Salpingo-oöphorohysterectomy was performed. Examination of the removed specimens showed the two rounded uterine masses attached below by a band of fibrous tissue. Careful examination failed to show a cervical canal in either mass. The right mass is decidedly the larger, measuring 5 x 3 x 3 cm.

The right Fallopian tube is so convoluted as to measure only about 3.5 cm. in length. The fimbriæ were adherent, and had attached a well-marked hydatid. The ovary is large, shows scars and small follicular cysts.

The mass to the left measures but 3.5 x 2 x 2.1 cm. The left tube is also convoluted, measures about 8.5 cm. in length, the outer 4.5 cm. being attached to a parovarian cyst measuring 4 x 3 cm. The fimbriæ are free. The left ovary is about one-third the size of the right, measuring about 2 cm. in length.

The organs are free from adhesions. The presence of two distinct rudimentary uterine bodies, united at their inferior extremity by a very rudimentary cervix, seems to justify the term under the classification of Kussmaul of uterus bicornis unicollis. *March* 11, 1897.

13. *Spindle-celled sarcoma of the ovary.*

Presented by Joseph McFarland, M.D., and Wm. W. Babcock, Jr., M.D.

Clinical history. Mrs. X., aged thirty-five years, married, housewife; menstruated from the time she was twelve until she was twenty years of age, regularly, freely, and without pain. At twenty periods of amenorrhea, lasting from six weeks to six months, developed, while a hard mass the size of a goose-egg was noticed in the left groin, which was movable and not painful. The patient married, and four years later the menses became regular, and an apparently normal pregnancy ensued. Six months after labor the menses returned with regularity and without pain, while the tumor could no longer be felt. When thirty-two years of age a period of amenorrhea began to develop, while a mass was again found, as before, in the left groin. It had the size of a hen's egg, but grew, slowly at first, then during the last six months rapidly, until it finally occupied most of the abdominal cavity and extended up under the costal margins. To the touch it was firm, hard, and bosselated; not tender on ordinary pressure, although blows caused agonizing pain.

During the past two years the patient has lost about thirty pounds in weight.

There were no urinary or rectal symptoms.

Physical examination showed a firm, bosselated tumor, filling most of the pelvis and abdomen. This was considered by most examiners to be a uterine-fibromyoma.

Abdominal section was performed January 25, 1897, by Dr. Charles P. Noble, through whose courtesy this specimen is presented. On opening the abdomen the amount of free fluid in the abdominal cavity was not sufficient to attract attention. The adhesions were not as a rule dense, and the entire tumor, with supravaginal uterus and appendages, was removed.

Macroscopy. The removed mass has a pinkish-gray color, the surface is bosselated, and there is a distinct capsule supporting numerous large, tortuous bloodvessels.

On the posterior surface the capsule appears to be thickened into a broad median raphé.

Over the surface are numerous clear-fluid, thin-walled cysts, a few centimeters in diameter. In consistence the mass is soft, semifluctuat-

ing, and this softness, together with the color, the rounded outline, the bosselation, and the tortuous vessels, causes it to bear a decided resemblance to the cerebrum.

Section of the neoplasm was not made, but it is supposed that the interior contains cysts.

In outline it consists of a larger, rather flattened, spherical portion, which occupied the abdomen, united to a smaller spherical portion which exhibits the moulding of the pelvis. Across the anterior surface of this pelvic portion are suspended the uterus and its appendages. The tumor proper is seen to be an outgrowth from rather than a general enlargement of the right ovary. The uterus and left appendage were attached to the capsule by rather light adhesions. The long (vertical) diameter of the neoplasm is 24 cm.; the breadth of the abdominal portion is 23 cm., and of the pelvic portion 13 cm., while the general thickness is about 14 cm. The weight of the growth, with the attached uterus, is 3.8 kilos.

The uterus is small and normal in appearance, the supravaginal portion measuring 6.3 x 5 x 3.2 cm.

The right Fallopian tube is 6.3 cm. long, and, except for a moderate enlargement of the distal portion, it shows no marked gross lesion. The left Fallopian tube is 10 cm. in length, and has a diameter of about 1 cm. near its outer part. Attached to one of the fimbriæ is a clear-fluid cyst, about 2 cm. in diameter. Both sets of fimbriæ are free. While the left ovary is large and cicatricial, it shows no gross evidence of a new growth.

Microscopy. The specimen was hardened in 10 per cent. formalin-solution, and sections were made from what appeared to be the original beginning of the growth at the site of the right ovary, and also from the abdominal portion of the tumor. The former sections, on staining, were found to consist of two portions, one, corresponding apparently to the outer portion of the growth consisting of fibroconnective tissue, resembling that found in the ovary. The other, or deeper portion, consisted of a mass of spindle cells, rather irregular in size and grouping, except for a tendency to arrange themselves in whorls. It was suggested that these whorls had formed around the vascular or lymphatic channels of the neoplasm. Erythrocytes, however, were not noted in their interior.

The nuclei of these spindle-cells were rather large and rounded. Anuclear, faintly granular areas suggested the presence of a rather widespread mucoid degeneration. Vascular spaces were fairly numerous.

Sections made from the abdominal portion of the growth showed a structure similar to that last described.

No Graafian follicles were found in any of the sections.
The diagnosis was spindle-celled sarcoma of the right ovary.
At this early date no tendency toward recurrence has been noted.

Discussion.—Dr. Beyea said he had seen four instances of sarcoma of the ovary. All were symmetric, and took the shape of the ovary. He had not seen a case in which the tumor grew from one end like the present one. *March* 11, 1897.

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14. *An hypertrophied prostate gland removed from a man seventy-eight years old six days after castration.*

Exhibited by Joseph P. Tunis, M.D.

The specimen which I am about to show you was removed from a patient of mine at the Methodist Episcopal Hospital. A detailed

Section of bladder and hypertrophied prostate of Mr. A. The nodule which prevented the entrance of the catheter is shown cut across.

report of the case has appeared in the *International Clinics*, January, 1897, vol. iv. sixth series, p. 202. The patient was seventy-eight years old, and had suffered for ten days previous to admission from retention of urine. Every form of catheter having failed to draw off the urine, he was tapped suprapubically under local anesthesia by chlorid of ethyl, and a quart of urine drawn off. The bladder was then washed out with boric acid solution and the canula left in situ. The patient experienced so much relief from this operation that he persistently refused to give his consent to the more radical operation of castration. Despite the free drainage from the bladder, the patient developed uremic coma two or three times, and was brought out of these attacks only by the frequent application of the hot pack combined with saline purgatives and diuretics. His urine showed unmistakable evidence of chronic interstitial nephritis.

The suprapubic exit for the urine failing, he finally gave his consent to castration, and I removed both testicles under Schleich's anesthetic. He suffered no pain from the operation, and the following day I was able to introduce a prostatic catheter into his bladder without any difficulty. This same instrument had been tried every previous day without success. He died six days later of uremic coma, and the kidneys confirmed the diagnosis which had been made from the urine. There was evident cystitis and pyelitis also. The accompanying photograph shows admirably the enormous hypertrophy of the gland, and the microscopic sections show the excessive amount of connective tissue which was present. *April* 8, 1897.

15. *Congenital anomalies of the kidney.*

Exhibited by A. Hand, Jr., M.D.

This series of kidneys illustrates the congenital anomalies found at the postmortem table of the Children's Hospital during the past year. They all have one point in common: that they were associated in each case with some other malformation in another part of the body.

This left kidney, which has been shown at a previous meeting, is the seat of what might be called compensatory hypertrophy, there being a congenital absence of the right kidney. The associated malformation was a meningocele in the lumbosacral region.

These horseshoe-kidneys were taken, the one from a case of imperforate rectum and hypospadia; the other from a boy, aged nine years,

who died with a sarcoma of the anterior mediastinum, which had given metastasis to the heart, and, as you see, to the kidney. The congenital malformation in this case is of interest in connection with the theory of the embryonic origin of tumors. *May* 27, 1897.

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16. *A case of renal tuberculosis.*

Exhibited by J. Dutton Steele, M.D.

The following case occurred in the wards of Dr. Joseph Hearn in the Philadelphia Hospital. I am indebted to him for the clinical history.

L. H., aged twenty-three years, a native of Ireland, white, married. There is no tuberculous family-history. She was admitted in October, 1896. The patient had measles in childhood; influenza five years ago, which left her with a "weak right side." In January, 1896, she was delivered of a six-months' fetus, and since that time has been confined to bed continually. She has become much emaciated and has a profuse diarrhea. Examination of abdomen shows a large mass the size of a baby's head in the right lumbar region. This is movable, and the skin is not adherent. The tumor is not painful except on deep pressure. A slightly tympanic note is heard in percussion over this mass. There is marked edema posteriorly in the lumbar region on the right side, extending up along the thoracic wall. The tumor appears to be distinct from the liver. There is no jaundice. There is no pain in the lumbar region. Micturition is frequent and sometimes painful. Urine: albumin three-fourths by volume. There is much pus, no casts, and no blood.

She was transferred to the surgical wards for operation, but this was deemed inadvisable. Her temperature then was 101°, but soon fell to normal, and remained subnormal until death. Her breathing was so shallow that physical examination was impossible. Now complains of some pain in the tumor. Is having many large, green liquid stools. Examination of urine just before death showed albumin, pus, casts, but no blood. She died October 26th.

The autopsy was made by the writer at the Philadelphia Hospital twenty hours after death. The peritoneal cavity is normal, except that the liver is pushed forward and barely reaches to the costal margin. This is caused by a large tumor lying behind the peritoneum in the right lumbar region. The pleural sacs each contain a small amount of clear fluid. There are thickening of the pleura and adhesions at each

apex. The upper lobes of both lungs show considerable consolidation at the upper portion. This is apparently due to a pneumonic process, with scattered areas of caseous degeneration, especially about the bronchi. Considerable pus flows from these tubes on section, and they contain caseous plugs. At the apex of the right lung is a cavity filled with purulent material. The left kidney is swollen and tense. The capsule strips readily, the cortex is swollen and of a green color, and the pyramids are congested. The pelvis and ureter are normal. The right kidney is found to constitute the tumor mentioned above. It is 25 cm. in length, 18 cm. broad, and 15 cm. thick. The organ is surrounded by a considerable amount of fat that appears normal. On section the kidney is found to be extensively degenerated. The capsule is thickened and firmly adherent. The cortex measures 3 cm. in width, is dense and hard and of a chalky white color, with numerous spots of extensive cheesy degeneration. The pyramids are represented by cone-shaped masses, with central cores of cheesy material which empty into the pelvis, which is filled with pus and caseous débris. The walls of the cones are dense and white; the walls of the pelvis are extensively infiltrated, and its mucous membrane is ulcerated.

Microscopic examination of the right kidney showed hardly a trace of kidney substance. The capsule was greatly thickened and there was an increase of connective tissue in the walls of the masses that represented the pyramids. Around the margin of the degenerated area were collections of round and epithelioidal cells and occasionally giant-cells. A number of sections were stained for the tubercle-bacillus, but without success. However, the diagnosis of tuberculosis was made upon the characteristic picture presented by the collection of cells associated with the extensive degeneration.

The wall of the ureter is also much thickened. This infiltration extends downward along the whole length of the tube and involves the bladder wall around the orifice of the ureter. The epithelium from the pelvis to the bladder is degenerated and replaced by caseous material, but the lumen does not seem to be obstructed. The epithelium of the bladder has apparently entirely escaped the inflammatory process.

The external genitals are perfectly normal. The lymphoid tissue throughout the body is apparently entirely normal. Nothing else of interest is found in the body.

Section of the left kidney revealed much fatty degeneration and chronic parenchymatous nephritis.

The case reported belongs to the chronic variety of renal tuberculosis. The sequence of events is doubtful, and the case might reasonably be

regarded as one of the rare instances of renal tuberculosis secondary to cryptogenic infection through the mucous membrane of the intestinal or respiratory tract. However, the adhesions at both apices, and the cavity on the right side, together with the fact that the lesions of the lung are of the type of an infection by inhalation, suggest that probably the disease was primary in the lung, and that the older lesion was masked by a sudden extension of the process about it at a comparatively late period, and shortly before death.

The histologic portion of the above report is from the Pepper Laboratory of Clinical Medicine. *June* 10, 1897.

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17. *Kidney with double ureter.*

Presented by FRANK WHITE, M.D.

J. S., a low-grade imbecile, admitted to the Pennsylvania Training School for Feeble-minded Children, July 7, 1892. He had had all the ordinary diseases of childhood. He had not received much treatment, except for a few minor troubles, since his admission to the institution. On April 5, 1897, a small carbuncle started at the lower angle of his mouth on the left side. The usual treatment was applied for the first twenty-four hours, the carbuncular lesion was freely incised, pus evacuated, and the lip irrigated with an antiseptic solution. Any attempt at treating this carbuncle would be followed with such profound shock that it necessitated several hours for his reaction. The next day, the 7th, the lip and chin were improved, but there was some enlargement of the glands of the face and neck on the left side. The boy was dull; temperature 100° F., but he did not complain of pain. The night of the 7th the boy had some fever and was delirious. Throughout the day of April 8th his temperature ranged between 103° and 105° F. He had very active delirium, necessitating restraint in order to keep him in bed; vessels of the eyes were much enlarged; muscles of the neck rigid. This condition continued, with other complications, until April 11th, when he died.

The *postmortem* was made twelve hours after death. A well-nourished boy. Rigor mortis marked. Four small incisions found on the left side of the face. Some postmortem lividity. Scalp thick and filled with blood; skull remarkably thin throughout. Meninges on left side inflamed, also some cerebritis along anterior temporal convolutions. *Thorax.* Engorgement of right lung; acute pleurisy of the left lung. *Heart.* Acute pericarditis, with about three ounces of fluid in the pericardium. Liver enlarged, and filled with venous blood. Spleen en-

larged, very soft and friable. Nothing abnormal was found in the gastro-intestinal tract. The right kidney was engorged, but otherwise normal. The left kidney presented the following points of interest: Numerous congenital fissures were found running throughout the entire convexity, some running longitudinally, others transversely to the long axis of the kidney. Probably two of the largest were found on the posterior surface and extending into the hilum. Numerous small depressions were also found in this region. The uppermost part of the kidney was thick and blunted, while the lowermost was flat and tapering. The cortical portion was probably no thicker than would be expected from a kidney of this size. The medullary portion was proportionately enlarged. The renal artery of the main hilum was almost twice the size of the average renal artery. The vein would seem to be almost normal in size. In the second hilum the artery was small, the vein was also small; the two ureters connected with two separate pelves. Each pelvis was found to be almost as large as in the normal kidney. Length of ureters nine inches, running entirely separately until within one inch of the bladder, when they formed one common duct. Bladder dilated, mucous membrane thickened. Probably the most interesting point of this case was the fact that so many congenital anomalies were found, and all were on the left side. First, a double great toe; second, the webbed condition of the second and third toes: third, double ureter of the kidney; fourth, supernumerary finger attached to the left side of the hand, but which had been removed in early life.

Weight of various organs: Brain, 1356 grams; left lung, 485 grams; right lung, 513 grams; heart, 220 grams; liver, 1617 grams; spleen, 279 grams; right kidney, 117 grams; left kidney, 195 grams.

June 10, 1897.

VI. THE NERVOUS SYSTEM.

1. *Ricin-poisoning: Experimental lesions produced by the action of ricin on the cortical nerve cell of the guinea-pig's and rabbit's brain.*

Presented by Henry J. Berkley. M.D.
JOHNS HOPKINS HOSPITAL, BALTIMORE.

The Effect of Acute Ricin-Poisoning. Since the completion of the last article of this series on the condition of the nerve-cell in serum-poisoning, two papers by Monti have appeared, showing lesions somewhat on the same order as those we have seen in chronic alcoholism and serum-poisoning, but in each separate one there is sufficient difference in the form and general deportment of the tumefactions, as well as in the relation of the gemmulæ to the morbid process, to enable an accurate observer to differentiate without difficulty in a well-stained slide from what pathologic condition it was prepared.

Monti in his last article devotes a portion of a page to the gemmulæ, the first detailed notice of these important parts of the neuron that I have met with. He designates them as spines, describing them as having *en masse* the appearance of a bunch of moss covering the protoplasmic stem, and mentions that they are constantly to be found in the cells of the adult brain, the latter statement agreeing with what we have already determined.

A further consideration of the gemmulæ may not be out of place, and has a future bearing on the pathologic conditions hereinafter described. In slides prepared according to the long Golgi method I have been unable to determine them with any constancy, and even with the ordinary osmium-bichromate fixing method of Cajal they are apt to be inconstant, at least in my experience. With the silver phosphomolybdate method they are constantly stained, are of fixed shape, according to the variety of cell from which they proceed, and are present on every kind of nerve-cell in the cerebrum and cerebellum in varying numbers, but reach their highest development on the psychical and Purkinje cells.

Opportunity has hitherto not been afforded me to study their development after the beginning of extrauterine life, but judging from works of others, they do not seem to be present on the ganglion-cells of the cortex until sometime after birth, certainly some weeks in the lower

animals (van Gehuchten, etc.). They are present on the protoplasmic prolongations of the pyramidal cortical cells (Cajal) of all vertebrates, but their deportment and expansion depend greatly on the animal from which the section is taken. Generally speaking, they are less prominent in the lower vertebrates, and reach their highest development in man. Even in the human being their numbers and luxuriance depend on the individual, for in persons of low mental grade they are not nearly so prominent as in better endowed individuals. Their development appears to depend largely on the receptivity of the species of animal to education from external impressions, dull vertebrates like rana presenting a wide difference from animals capable of receiving a higher education, as the dog; hence we are inclined to believe that there are certain inherent qualities in the development of the cerebral cells themselves that render one species of animal more capable of receiving and retaining impressions from extraneous sources than others; in other terms, they have memory for past events, and not only is the quality of the protoplasm different in one animal from another, but there are differences in the actual construction of the neuron itself.

Moreover, each psychical cell of the cortex possesses an individuality of its own, the shapes and general appearance of the cells differ widely, the general arrangement of the principal dendrites is different, the disposition of the gemmulæ varies slightly between each cell, the apical protoplasmic extensions are sometimes wide-spreading, sometimes do not expand into a number of branches until they reach the neighborhood of important nerve-endings in certain well-defined regions of the brain-rind.

A more extensive examination of histologic specimens leads me to reopen and reconsider the question of the nodosities on the dendrites being normal, or artifacts from the deposit of fine precipitate on the dendrons giving them the appearance of being covered with nodes, as is the view taken by Flatau (*Arch. f. mik. Anat.*, Bd. 44).

In early postfetal life the dendrites are covered with varicose swellings that may persist over a variable time. In some adult animals these varicosities seem entirely to disappear (man, rabbit), while in others they are still persistent, though in very inconsiderable numbers (guinea-pig). Even when they are present in these animals they are always of very small size, present rather the appearance of irregularities than varicosities along the finest stems, especially toward their tips, and are covered, equally with the other portions of the stems, with the gemmulæ, and never encroach upon the thicker portions of the primordial or basal dendrites. That they are not artifacts is further evidenced

by the fact that under intense illumination they show none of the characteristic appearance of precipitate, but fine parallel lines converging on the unswollen portion of the dendron. Differences in the staining qualities of the cells are very much more puzzling than any deposit of precipitate, and may occasionally lead to wrong interpretations.

All of our specimens were placed in the fixative fluid almost immediately after death, and accordingly I consider that we are able to exclude even the suggestion of postmortem changes without further comment on this side question.

Hitherto in these studies we have had the absence of the gemmulæ from the swellings to aid us in determining whether we were observing a normal or abnormal nodule on a dendrite. In the present investigation, especially in the animals that lived but a short time after the injection of the poison, we no longer have this constant guide; for in the short space of a day and a half it could hardly be supposed that there would be complete destruction of these most delicate portions of the nerve-body; and such, indeed, is the fact; though we do meet with a certain diminution of the pin-like attachments to the protoplasmic stems. To counterbalance this loss of control we find in the cases of pathologic swelling the lateral buds pushed further apart from each other than is natural, and while often they may appear to be present in diminished numbers, an actual count will show that they closely approximate the numbers on the normal dendrites, though at times, when the swellings have considerably advanced, they perceptibly diminish. This condition is, however, only in the very earliest stages, when alterations in the protoplasm have hardly begun; in further advanced stems they gradually disappear, until when the thicker portion of the primordial dendrite becomes involved in the swelling, which is never the case in normal preparations, they vanish rapidly. This retention of the gemmules is a strong argument against the tumefactions being artifacts, and clearly shows that the swelling comes from within the substance of the stem and pushes the gemmulæ, which are still adherent, outwardly and apart.

The researches upon the action of ricin, the amorphous poison obtained from the castor-bean by Ehrlich and Flexner, induced me, after reading the paper by the last-named writer, to infer that this agent would be an excellent one to cause pathologic alterations in the protoplasm of the ganglionic nerve-cells.

Dr. Flexner very kindly placed at my disposal the brains from a number of his ricin-rabbits and guinea-pigs, all fixed in Müller's fluid.

I cite, in abridgement, the lesions discovered by Flexner in the acute

cases in tissues other than the nervous system: The lymph-glands are everywhere throughout the body swollen and softened. The small intestines are distended and pale, their contents being glutinous, soft, grayish-white in color, and resembling in appearance cholera-stools. The patches of Peyer in both the small and large intestines are elevated, swollen, and pale. The heart is filled with blood, the right ventricle especially being distended. The blood is fluid, or soft clots may appear. Neither the kidneys nor the lungs show especial alteration. The spleen is slightly swollen and softened; the liver is large, soft, and yellow in color.

The microscopic pathologic alterations found in the viscera in the acute cases of poisoning are very profound. In the lymph-glands hemorrhages are commonly present, and many of the lymphatic elements are necrotic, and fragmentation of the nuclei is common. The lymphatic apparatus of the intestine shows, in an analogous manner, the destructive influence of the poison, but cell-death is much more extensive, and the swelling of the follicles is clearly due to the increase in the number of the lymphoid elements. The destruction is, however, not limited to the lymphatic elements in the follicles, but those in the villi suffer extensively, and the epithelial elements are little spared. The spleen shows changes similar to the lymph-glands, and these are localized in part in the Malpighian bodies.

The liver presents the greatest variety of forms of cellular death. In the capillaries the endothelial are sometimes fragmented and necrotic, and the leukocytes suffer a similar change. But in the liver-cells the process reaches its height. The yellowish areas visible to the naked eye correspond to foci of coagulation necrosis of liver-cells. The liver-cells in some of these areas are still preserved, but they are hyaline in appearance and devoid of nuclei, while in others the cells are much altered in appearance and staining properties, and the nuclei have undergone another form of necrosis. They have died as a whole, becoming paler and paler, until they finally refuse to stain, with apparently a synchronous alteration of the cellular protoplasm.

These foci of necrosis are often surrounded by large quantities of granular detritus. Some of the detritus must have come from emigrated cells, as we now know happens in these necrotic foci, where they suffer the same fate as the tissue-elements.

The lymphatics in the interlobular spaces of the liver are choked with the nuclear detritus. Manifestly such an amount of nuclear material could not come from the cells lining the lymphatics nor from the cells of the lymph. There is only one interpretation of this process possible:

it represents the nuclear detritus of the tissues and emigrated cells which had been swept by the lymph-vessels into the current.

As it is not possible that such extensive changes should occur in the other tissues of the body without implication of the nervous elements, we obtained from Dr. Flexner the brains from six of the acute cases, and after, hardening them carefully, further treated them after the silver phosphomolybdate method and several nuclear stains, as in former studies. For control material we used the fresh cerebra of a healthy full grown rabbit and a guinea-pig, treated according to the same procedure as the pathologic material, allowing them to remain exactly the same length of time in the hardening and staining media.

CONDENSED TABLE OF THE ACUTE RICIN-SERIES.

No. in Series.	Animal used.	Age.	Weight.	Date of inoculation.	Death occurred in hours.	Amount of ricin.	Clin. cause of death.	Gross anatomical lesions.	Remarks.
1	Guinea-pig.	Adult.	510 grams.	Aug. 31, 1895.	36	1 mg. Subcutaneously.	Intoxication.	Usual lesions.	All the animals received the inoculations subcutaneously, with the exception of the rabbit, and with the usual precautions to avoid infection from extraneous sources. The inoculation was only made once in each case.
2	"	"	680 grams.	Sept. 4, 1895.	40	0.5 mg. Subcutaneously.	"	Edema and hemorrhage at site of inoculation.	
3	"	"	740 grams.	Sept. 4, 1895.	48	0.25 mg. Subcutaneously.	"	Usual lesions.	
4	Rabbit.	"	1700 grams.	Aug. 30, 1895.	14	1 mg. Intravenous.	"	"	
5	Guinea-pig.	"	700 grams.	Sept. 8, 1895.	72	0.125 mg. Subcutaneously.	"	"	
6	"	"	450 grams.	Oct. 10, 1895.	37	1 mg. Subcutaneously.	"	"	

It will presently be seen in the description of the individual cases that in the five guinea-pigs the amount of destruction of the nerve elements depends not altogether on the amount of the poison injected into the system of the animal, but also on the duration of the poisoning. Almost equal changes can be produced in seventy-two hours by the inoculation of 0.125 milligram ricin as by the inoculation of a whole milligram lasting over a period of only thirty-six hours, and perhaps had the animal lived a still longer time the changes would have been even more pronounced. On the other hand, in the single rabbit subjected to an intravenous inoculation with 1 milligram ricin the

destruction of the protoplasm of the nerve-cells was greater than in any other case, though Dr. Flexner informs me that he has always noted greater intensity of the action of the toxin in intravenous than in subcutaneous injections.

The Effects of the Toxic Substance Acting Directly on the Substance of the Nerve-cell —(*a*) *Lesions seen by nuclear stains.* The study of the two cases selected (Nos. II. and IV.) for examination of the blood-channels' walls with nuclear dyes shows lesions entirely similar to those found by Flexner in the abdominal organs, only not of such intensity. In the finer vessels there are many places in which the endothelial nuclei are swollen, receive the stain badly or not at all, and are occasionally necrotic and fragmented. The perivascular channels contain considerable amounts of a fine-grained detritus that does not take up any other dye than eosin, and this only in sufficient quantities to give it a pale-rose tint. This detritus is to be found in the spaces around all the medium-sized and larger bloodvessels, and the amount of it is in places sufficient to retard the lymph-currents. All evidence of the presence of thrombi in the smaller vessels is absent, and the numbers of white blood-corpuscles within the vessels and perivascular spaces are inconsiderable.

The protoplasmic bodies of the majority of the nerve-cells appear to be unaffected by the poison, a few seem to be breaking up into granular matter. Very curious nucleolar figures are to be seen everywhere in the nerve-cells of the gray substance. The fine dust-like granules and the two or three larger particles that are normally present are no longer to be seen, but in their place in the interior of the nuclear ring lies a brightly-stained, irregular mass, sometimes of horseshoe outlines, or at others of varied shape, probably composed of the aggregations of the nucleoli and nucleolar dust.

(*b*) *Lesions seen by the silver phosphomolybdate method of staining.* The six cases divide themselves, so far as the intensity of the destructive influences determined by the drug are concerned, into two principal groups: those in which the pathologic alterations are limited to the finer branches of the cell, there obtaining fine swellings of the outermost portions of the protoplasm of the cell, accompanied by but little denudation of the gemmulæ, and the remaining portions of the cell are free from apparent injury; and, secondly, cases in which there is involvement of the dendrites down to the cell-body, with great disappearance of the lateral buds, and certain changes in the corpus of the cell.

The first detail is to be found after the continued action of the drug for comparatively short periods of time; the second, either after the

long-continued action of such small doses of the toxic substance as will enable the animal to survive a considerable number of hours, or, after throwing into the circulation of a proportionately very considerable amount to the weight of the animal of the toxin, inducing a sudden and intensely destructive effect upon the cerebral cells (Cases IV. and VI.).

All the cells of the cortex beside the fusiform and angular cells of the outer layer, which stain infrequently, are, irrespective of the anatomic situation of the different layers, involved in greater or less degree by the degenerative process; but as the pyramidal cells of the second are the most numerous, best defined, and important, we shall take them as the sole type of cortical cell in which degenerative changes are apparent, if only from the fact that their greater constancy in receiving full impregnations with the silver salt renders one less liable to be wrongly influenced by irregularities in the staining qualities.

In the first class we find on the tips of the dendrons, particularly those situated within the limits of the molecular layer, well-defined, rounded, irregular, and fusiform swellings, which appear to begin toward the extreme outer endings of the protoplasm and gradually extend downward. On these tumefactions the gemmulæ may or may not be involved. When the swelling looks comparatively recent the gemmulæ do not seem to be greatly diminished in numbers, but when they are older they disappear. That the swelling commences in the substance of the dendritic stem is evidenced by the circumstance that the lateral buds are pushed aside at first before they begin to atrophy and disappear, and then undergo a change that is not well defined by the silver-impregnations. They absorb less of the staining material, are yellowish in color instead of black, and then, before any other definite alteration takes place, they fall off the stem, and presently the detritus is taken up by the lymphatics of the brain and they vanish entirely. As the diameter of the gemmule does not exceed one-tenth that of a red blood-corpuscle, it is quite easy to understand that they may be very rapidly absorbed by the lymphatic apparatus and quickly lost. It is not uncommon to see a few of them lying in the tissue close to the side of the swollen stem, but without any visible connection with it, and from the extreme tenuity of their attachments to the protoplasm of the stem and the dependence upon it for nourishment it may be readily understood that the slightest injury affecting the vitality of the parent dendrite will instantly cause an involvement of the side buds of the branches. When the swellings become fairly numerous along the finer dendrites, the stripping off of the buds soon becomes very apparent.

In these earlier cases there is little atrophy of the stem in the intervals

between the tumefactions, the gemmulæ being retained in but slightly diminished numbers and showing only occasional variations in the staining. The thicker dendrites are in the first class rarely, if ever, involved, nor does the cell body lose any of the definite sharpness of contour or appear swollen or malformed.

The basal dendrites seldom participate to any extent in the first steps of the tumefactive process, though one is occasionally seen with a small swelling along the course of one of the branches; but this is exceptional.

The axons from all the cortical cells display none of the irregularities in their outlines or abnormal increase in the number of varicosities that have been described in the more chronic diseases. Likewise the collaterals are equally finely contoured, and the bulbous endings situated upon short lateral branches arising from the principal fibers are all well stained, and have every appearance of being healthy.

Fibers coming from the deeper layers, axons from the cells of Martinotti, diffuse meshworks from the intermediary cells are all well impregnated and present no difference from those in control preparations.

Neurogliar changes seem in the earlier cases to be absent. It is true that the staining, in the first series of sections, of the different varieties of support-cells was not very good, only scattered ones being visible occasionally in the field, but those stained presented every appearance of health.

No coarse lesions were visible in the walls of the bloodvessels in Cases I., II., and III., with the exception of an inconsiderable number of perivascular extravasations in the second case. Even in the hemorrhages the blood-corpuscles were perfectly preserved, showing that they took place about the time of the exitus.

In the cerebellum, in the first class of cases, we have met with but imperfect staining of the nervous and support-elements, the Purkinje cells and Bergmann fibers excepted.

The aspect of some of the last-named nerve-cells is exceedingly instructive. In the first place they present the usual mixture of perfectly normal and damaged cells in a similar manner to the cellular layers of the cerebrum. A damaged cell may have some of its branches entirely sound, while others have lost the greater number of their gemmulæ, and have very ragged outlines. There is now very little irregular tumefaction of the protoplasm of the stems, but on the other hand an apparent thinning, seemingly due to the stripping off of the gemmulæ.

An occasional basket-cell with its peculiar axon is now and then well stained; some of them show alterations of the dendrons, some do not do

so, but the nerve-extension is always normal. In the molecular layer nothing is stained beyond certain nerve-fibers, and in the central core of the leaflet, in addition to these, a few short-rayed neuroglia-cells. The Bergmann support-cells of the molecular layer afforded from their constant staining opportunity for accurate investigation of their structures, but gave no indication of any degenerative process affecting them.

The changes found in the nerve-cells of the second class of cases are relatively much more interesting from the greater intensity of the alterations found, as well as the absolute pathologic significance they possess. It is always to be remembered, however, that except in one case (No. 6) there was nothing approaching an involvement of all the neurons of the cortex, but that actually a large proportion of cells in all portions of the layers has every indication of health, and while very extensive alterations are found in very many of the neurons, those that are normal and those that belong to the first class greatly outnumber them.

In every section taken from Cases IV., V., and VI., the neuron is practically reduced to the main apical dendrite, with sometimes a few of the basal ones remaining intact, sometimes they are degenerated. The apical dendrite is shorn entirely of the lateral branches, or they are reduced to irregular filaments, the outspreading uppermost branches vanishing completely. The tree like form has departed, it is reduced to a trunk ill-shapen and knotted, and its roots are dying. Along the stem of the trunk we find swellings of various sizes and shapes; within the interval between the swollen portions, lines of protoplasm much thinner than at corresponding points in normal specimens. When we look for the gemmulæ we find they are still present in some places, in others they are entirely absent, but as a whole their numbers are greatly diminished, the remaining ones ofttimes appearing to be badly stained. In the most advanced of these instances the cell-body appears not quite as angular as in the control-specimens, but otherwise there is no definite alteration.

Turning from these middle forms to others that have suffered more considerably in the atrophic process, we find an extensive reduction in the length of the apical and basal dendrons. Some of these are thinned to a great degree, but have few tumefactions, but others have numerous swellings of all sizes, both in the apical and basal dendrites, the primary ones always showing the greater amount of tumefaction and atrophy. There is still retention of very limited numbers of gemmulæ. Quite often, however, there is entire absence of them over certain of the stems, which now appear thinned to fine filaments, with the remains of a nodule showing at intervals.

In the most degenerated type the basal dendrites are reduced to stumps, the primitive one is atrophied down nearly to the body of the cell, is mainly formed of a few large tumefactions irregular in shape, and the gemmules have utterly vanished. The corpus of the cell, previously angular, is now seen as a rounded body with imperfect dendrons extending from it, but there is as yet no breaking up of the substance of the cell. The destruction of the receptive function of such a cell must necessarily be complete.

This concludes the description of all the positive changes in the nerve-cell from the effects of acute ricin-poisoning. There are, however, a few cells whose appearance merits a short notice. These nerve-bodies have the aspect of those described by Monti in his work on inanition, but in the present instance we are inclined to regard them as owing to defective staining. The cell-body is well preserved, but at a comparatively short distance from the corpus the branches begin rapidly to attenuate and assume a beaded character, with the portions intervening between the beads so thinned and ill-stained as to be apparent only on close examination. The fact that we find an entire absence of the gemmulæ everywhere, on both thicker and thinner portions of the dendrites, which is not usually the case except in far degenerated cells, leads me to infer that they are cells difficult to impregnate by the silver-salt, and in which the staining has only begun in the protoplasmic extensions.

Lesions of the neuraxons of the different varieties of the cortex have been so rarely found that they may be excluded.

Alterations in the neuroglia, on the other hand, demand more than a passing attention. In the last three specimens the staining of the support-elements was equal to any in the control-preparations, but apart from the peculiar local cells along the subpial margin there is but one form of neuroglia-cell impregnated in either medullated or gray tissues, and these are the so-called mossy vascular cells. I do not consider this absence of the star-rayed cells as important, having met with the same peculiarity in staining in many control-preparations, but we find certain differences between these in the ricin cases and those in the control.

There is no either relative or absolute increase in the numbers of the cells, indeed, they are in rather fewer than normal proportions, but certain qualities in their bodies attract attention. They are universally much larger than the control, are apparently swollen, even globular in outline. The extensions, too, are thicker and more nodular, though they do not cover more territory than customary. Are these elements, which belong to the lymphatic apparatus, taking up the detritus from the degenerating protoplasm of the nerve-cells and becoming engorged?

In Cases IV. and VI. there was almost perfect impregnation of all the elements of the cerebellar layers, and the usual mixture of normal and abnormal cells, fewer of the former for the first time in Case VI. than in any preceding instance. Now the destruction of the protoplasm of the thicker and thinner branches is uniform and profound; they are entirely stripped of their buds, are irregular and gnarled, and the upper branches of the cells are entirely atrophied, and have in part disappeared.

Equally extensive changes have taken place in the intermediary and small cells of the molecular layer, but with all the destruction the neuraxons have not become involved, and especially beautiful staining of the branching axons of the intermediary and the peculiar nervous prolongations of the basket-cells were obtained to their terminations.

The staining of the neuroglia is also nearly perfect, but in contradistinction to the staining of the same elements in the cerebrum, the star-rayed neuroglia-cells, especially in the white tissues, are numerously impregnated, but exhibit nothing of import, while the short irregularly rayed cells, though in the minority, give evidence of swelling of the protoplasm of the bodies, equivalent to that in the cerebral cortex. The fibers of Bergmann, belonging solely to the support-type of cell, have no evidence of implication in the degenerative process.

Conclusions.—The poison ricin, whose action is in many ways similar to that of many toxalbumins of bacterial source, is capable of exerting a deep and extensive degenerative influence on the protoplasm of the nerve-cells of the brain. This action of the drug can, probably, be explained in two ways: its direct influence on the protoplasm of the cell, after having been taken up by the bloodstream, starts disturbed cellular metamorphosis; and, secondly, the nutrition of the cell is directly influenced by the disturbance of the blood-supply, by reason of changes in the walls of the smaller vessels, which conduce to and augment the direct action of the poison. Overwhelming destruction of the cellular protoplasm, rendering the lymphatic apparatus unequal to cope with and remove the detritus of the cells, may also become an important factor, as the more products of the dead tissues that remain in close proximity to the living ones, the more rapid, presumably, is the destruction of the remaining living elements.

The action of the drug is proportionate to the amount of it injected into the body of the animal, also to the duration of the poisonous action before death occurs. Limited quantities continued over a considerable time exercise the same destructive action as more extensive doses acting during a few hours.

The fine extensions of the cells are first destroyed, later the thicker

ones, lastly the cell-body and axon, both of which resist the process for a long period.

The question of regeneration of the nerve-elements in animals that may survive the deleterious influence of a poison is of the first importance from a clinical stand-point in the future history of patients suffering from severe infectious diseases, but of this we have at present no data. We are, however, led to expect from the history of such cases in the human being that there is at least a partial regeneration of the nerve elements, though in such instances as I have had the opportunity of examining the recovery is far from complete.

Special Description of the Separate Cases.—No. I. Guinea-pig weighing 510 grams, inoculated with 00.1 mg. ricin, died in thirty-six hours. The brain of this animal did not harden well in the preservative fluid, and it was therefore discarded.

No. II. Guinea-pig weighing 680 grams, inoculated with 0.5 mg. ricin, died in thirty-six hours. There are no coarse vascular lesions visible in the silver-slides. The disks of the red blood-corpuscles, except in a few instances, are distinct and unchanged. In the capillaries are single rows of the blood-corpuscles very closely packed together; everywhere there is evidence of antemortem hyperemia. In two sections, single, small, perivascular hemorrhages were found at about the juncture of the molecular and second layer of the cortex. In every region examined there is evidence of turgescence and congestion of the smaller arteries and veins.

Definite lesions of the neurons are not extensive, and relate almost entirely to the parenchyma of the primitive and basal dendrites. The most common alteration found is that the lateral branches of the pyramidal cells within the molecular layer are studded with considerable numbers of swellings of various sizes and shapes, but without any considerable diminution in the numbers of the lateral buds. Occasionally a whole branch is so studded with these tumefactions as to form a striking object under low power, but this is rare.

In a considerable number of the cells the tumefactions are not confined to the finer dendrons within the molecular layer, but spread downward toward the lower portions of the primordial process, and such cells may be found in sparse numbers in all portions of the gray layers. When the stem of the primitive dendron is affected in this manner there is an evident though slight decrease in the number of the gemmules, and also a more apparent change in the disposition of the finer branches coming off from it; they seem to be less numerous than normally, and

in those that remain the lateral buds are less luxuriant than usual, though they still stain fairly well and possess all the attributes of the normal buds, except numbers. The swellings on the primitive or basal dendrites have the buds present, but they are usually more widely separated than on the natural stem, which is, presumably, accounted for by the tumefaction of the substance of the dendron pushing them more widely apart. As already stated, it is customary to find a distinct diminution of the buds on much-swollen dendrons.

The lower portion of the main apical dendrite is never involved in this case; indeed, lesions of the principal branches anywhere in the vicinity of the cell-body are extremely rare. Decided alterations of the corpora are entirely absent. Variations from the normal in the axons were not made out, though the staining of this portion of the neuron with the collaterals was often superb.

The tendency everywhere is for the altered cells to be scattered at intervals with large numbers of normal ones in between them; large areas of a section may be gone over without finding any traces of a diseased cell, then numbers of altered ones are seen among the normal; altogether the proportion of diseased neurons in the second case is by no means numerous. There are no distinct lesions of the neuroglia-elements, though, possibly, some swelling of the bodies of the vascular cells may be found.

In the cerebellum there is distinct loss of the gemmulæ on the branches of some of the Purkinje cells, combined with more or less irregular swelling of the stems, but these alterations are not numerous. The corpora and axons remain intact. The various fibers coming to and fro from the granular layer have no alterations in them as far as they are impregnated. The granules do not stain at all.

No. III. Guinea-pig, weighing 740 grams, inoculated with 0.25 mg. ricin; died in forty-eight hours. Equally with Case II., there is nothing of considerable interest concerning the larger bloodvessels or their contents beyond the fact that there is a certain degree of hyperemia of the entire rind of cerebrum and cerebellum, but nowhere is it sufficient to have occasioned great diapedesis of the blood-corpuscles.

The appearance of the nerve-elements does not differ materially from that of Case II., though the destruction of the branches of the dendrons is more frequent, all portions now being frequently involved, also the number of the primordial stems that are implicated in the destructive process is greater than in the other example.

The gemmules along the stems are somewhat sparser, and among those still retained there are some that are pale, ill-stained, and look as

if the morbid process had already begun in them. The cell-body is never frayed or eroded, yet the contours of the cells are slightly changed, as their angles, particularly those having the most damaged dendrons, are more rounded than is seen in normal preparations. Changes in the neuroglia-elements are, so far as those impregnated indicate, very slight in the cortex of the cerebrum and cerebellum.

In the last-named organ, the alterations in the Purkinje cells are on the same order as in the previous cases, namely, focal alterations of a few cells here and there, with the majority remaining intact.

There are no variations from the normal in the appearance of the fibers and their collaterals, nor are there distinct differences between the control neuroglia cells and those in this case. It is possible that the vascular cells are a little swollen, but the difference is uncertain.

No. IV. Rabbit, weighing 1700 grms, intravenous inoculation with 1 mg. ricin, died in fourteen hours. The bloodvessels in this case are far less congested than in those preceding it, the arterioles are empty as a rule, but the veins contain a small amount of blood, in which the red corpuscles are distinct, with their outlines perfectly preserved. The lesions of the blood-channels, as seen with nuclear stains, have been given in the first part of the article, for Cases IV. and VI.

The destruction of the protoplasmic branches of the cells is far greater than in any of the previous cases, and in many instances there is complete atrophy of all the branches nearly down to the cell-body, while in a host of others there is implication of the finer and coarser dendrites as well as the primordial stem. The loss of the gemmulæ is exceedingly great, many cells advanced in the degeneration only showing a half-dozen of them over the entire series of branches. The tumefactions are also larger and more numerous; altogether the process is more intense than anything we have met with in this series.

Besides a rounded appearance of their contours, the corpora of the cells show little alteration; one or two had what appeared to be an erosion near the nucleus and extending into it, but this is probably an artifact produced by the saw-like cutting action of the knife when the sections were made. Some of the dendrons of the several varieties of cells in the cortex show a beaded arrangement of the protoplasmic twigs, with complete loss of the lateral buds; literally every gemmule is absent from the dendrite, a different condition from anything we have before remarked in any of the sections. In these cells small varicosities appear at almost equal distances along the protoplasmic stem, which are tinged intensely black by the silver salt. In the intervals between the swellings the stem of the twig appears to have atrophied,

not only in the long direction of the stem, but also in its transverse diameter. Lateral branches seldom come off from these thinned twigs, and their whole appearance is indicative of an atrophic alteration in the protoplasm.

The question arose as to these cells being due to imperfect staining, as none of them are throughout equally well impregnated in the intervals between the beads, especially the small ones on the finer remaining stems; and the protoplasm is oftentimes almost untinged. These cells are to be seen among groups of well-stained cells, accordingly it is hardly possible that a single cell could be incompletely stained when others surrounding and overlapping it with their dendrons are well tinged both in body and dendrons.

In the cerebellum the alterations are very considerable, the gemmules are stripped off numbers of the Purkinje cells as an entirety, the branches are bare, but there are no large numbers of local swellings on the stems. The small cells of the molecular layer show modifications of the same changes, and their gemmules are not tinged in great numbers.

The nerve-fibers seem to be entirely unimpaired in any way, either in the cerebrum or cerebellum, both in respect to their continuity and change in the contour of the cylinders themselves. The collaterals, to their bulbous terminations, could often be traced, while some exceedingly beautiful examples of the outspread of the axons of the intermediary cells were found and traced to their endings, after large numbers of bifurcations. The *grimpante* fibers of Cajal in the cerebellum were particularly distinct in this specimen, as were likewise the axons of the Purkinje and basket-cells.

The alterations of the neuroglia are similar to those found in Case VI., and are described more in detail under that heading.

No. V. Guinea-pig, weighing 700 grams, inoculated with 0.125 mg. ricin, died in seventy-two hours. Inoculation, subcutaneous. While the action of the drug upon the protoplasm of the dendrites is not so grave as in the preceding instance (No. IV.), the lesions are of equal importance, marking the fact that not only does an overwhelming dose of the poison produce grave alterations of the protoplasmic substance, but that the effects of small doses continued over longer periods are equally injurious. The damaged cells are no longer scattered between comparatively normal ones, but are to be found everywhere, and the proportion of absolutely normal organs is reduced to a minimum, indeed this is so well marked that one has to observe right carefully to find an approximately normal cell. The basal dendrites are as equally impli-

cated as the apical, and many of them are also knobby and atrophic down to the thickened portions of the stems.

The lesions are, as an entirety, of the same general character as in the preceding instances, but there is less of the moniliform swelling of certain of the dendrons, with total absence of the gemmulæ, as was the case with No. IV.

The gemmulæ are accordingly retained in greater numbers on the swollen spots on the dendrons, while there is less thinning of the stems themselves. No cells were present with destruction of the corpora, but many were seen that certainly exhibited greater roundness of the bodily contour than is customarily found.

In the cerebellum the lesions are very well marked, the swellings of the stems and the loss of the gemmulæ are well shown, the small cells of the molecular layer and the Golgi cells of the granular have their prolongations swollen and irregular, and everywhere there are very decided changes.

The staining of the nerve fibers of all descriptions was in this case especially fine, and a careful study was made of them. In the cortex the axons of the projection-cells were finely tinged, very regular in contour, but an occasional varicosity showing at the juncture of a collateral or in the course of the fiber, yet nothing abnormal. The ascending fibers from the cells of Martinotti were traced to the outer layer without difficulty; the net-like arrangement of the axons of the intermediary cells was very distinct, and the branches could be traced to their bulbous knobs.

In the cerebellum the tingeing of the fibers was equally good, the *grimpante* fibers of Cajal were numerously stained, likewise were the axons from the basket-cells of Koelliker with the peculiar endings encircling the bodies of the Purkinje cells; also the axons from these last cells were well marked and were without varicosities.

The staining of the various forms of the neuroglia is similar to that in the last case. The vascular glia-cells are certainly swollen, the bodies and arms being more prominent than usual, but the other varieties have no evidence of morbid alteration.

No. VI. Guniea-pig, weighing 450 grams, inoculated subcutaneously with 1.0 mg. ricin; death occurred in thirty-seven hours. Naturally from the overwhelming dose of the poison injected into this small animal the lesions found were very extensive and widespread.

There is great destruction of the fine branches and thicker stems, with great numbers of the bead-like swellings stretching over all portions of the stems and branches of both the apical and basal prolongations.

Few swellings have any considerable size. The loss of the gemmulæ is very great, many of the injured cells have not a single bud upon them, while in others, and these are only present in small numbers, the gemmulæ are still visible, but are not equally or well stained. Even in the cells that retain something of the normal appearance it is very evident that the destruction of these delicate portions of the neuron has already begun, and that complete disappearance is only a question of a few hours' additional time.

In every portion of the cortex, and throughout every section that was examined, the same extensive destruction of the nerve-cell prolongations was found. Absolutely no alterations in the contour or general appearance of the corpora of the cells could be determined, not even the rounded outlines of the cells subjected to the longer action of the poison could be found, all the pyramidal cells and the other nerve-bodies retaining their usual angularity of contour and outline.

In the cortex the nerve-fibers, with the exception of the descending axons of the psychical cells, did not stain well. The latter were tinged to some distance from the cell-body, and exhibited no irregularity or other departure from the customary contours.

Differing from the first three of our cases, the neuroglia-structures were fully impregnated in all portions of the white and gray layers. Along the outer edges of the brain-rind the staining of the different varieties of support-cells peculiar to this region is almost perfect, while all through the cellular layers there is here and there, as in good histologic preparations, staining of isolated cells, but in contradistinction to these there is staining of only one variety, the vascular neuroglia-cell; no long or short-rayed cells appearing, even in the white tissues, where usually they alone are impregnated.

These vascular neuroglia-cells are large, the bodies seem of greater diameter than usual, are rounded, even globular in form, and the tentacles and short lateral projections are very coarse and distinct, seemingly swollen. The individual cells do not cover greater areas than is common among such cells. The reason why the short and long-rayed cells do not impregnate is of course unknown, but I have in exceptional instances found the same peculiarity in normal slides, and regarded it as being due to some defect in the hardening process, though in these normal preparations it is not usual to find the same numbers of vascular neuroglia-cells in the medullated tissues.

In the cerebellum there were, in accordance with the alterations in the cortex, similar ones in the Purkinje cells, with much destruction of the gemmulæ and swelling of the protoplasmic stems but these

changes are to some extent focal, or rather not so universally diffused as in the cerebrum. Numerous Golgi intermediary cells in the granular zone, basket-cells and small angular cells in the molecular layer were all well defined, and some exquisite examples of the terminations of the basket-cells were seen, but showed no alterations of their structures. The axons of the Purkinje cells only stained infrequently, and did not show any alterations. The resistance to the effects of the poison is therefore probably greater among the elements of the cerebellum than in the cerebral tissues. The peculiar bushy neuroglia-cells of the molecular layer only among all the neuroglia-elements of this region stained, but showed no ascertainable lesions either in the bodies or extensions.

The cerebral vessels showed very little congestion. The blood-corpuscles were perfectly preserved, in the capillaries there was a single row, not closely pressed together.

The Effect of Chronic Ricin-Poisoning on the Cortical Nerve-cell.—I have only been able to obtain a single brain from a case of chronic ricin-intoxication. The examination of this cerebrum was, however, very satisfactory and definite, and as it is at present impossible to obtain more material I have concluded to utilize the single case to complete this section of the series.

The 400 gram guinea-pig was inoculated by Dr. Flexner on September 11, 1895, with 0.03125 mg. ricin, subcutaneously. On the 18th day of the same month it received a like quantity of the poison, and on the 23d instant it received an additional one-eighth mg. Death took place on November 7, general emaciation being the best marked clinical feature of the course of the poisoning.

The principal anatomic differences between the acute and this chronic case lie in the size of the abnormal swellings upon the dendrites. Large numbers of small and medium-sized nodes are scattered upon the branches, but among them are some that have attained three or four times the size of the largest ones in the acute forms, and attract immediate attention. The vascular system does not appear to have suffered much in this case, there being no evidence of extensive congestions and dilatations of the perivascular spaces with exudation of the lymphoidal elements.

The staining of the nerve-elements and neurolgia was fair, and frequent tumefaction of the dendrons and loss of the gemmulæ were found, but the diseased cells were by no means as extensively distributed among the sound ones as in the acuter forms, and the proportion of the sound cells is vastly greater. It is perhaps only every third or fourth

dentrite in which we have definite departures from the normal. The tumefactions present the difference from the acute form in that the individual swellings are of greater size and the primordial stems have fewer of the fusiform tumefactions extending through the length of the dendrite.

Wherever there is swelling there is loss of the lateral buds in very pronounced degree, sometimes extending to complete ablation of these portions of the cell; at others a small number are retained adherent to the parent stem, but they do not fully receive the stain in the finer portions of the gemmule attached to the lateral aspect of the cell branch.

The stouter stems, near the cell-body, do not show any considerable variation from the normal. The corpora have nothing of the rounded swollen form noticeable in the cases in which death resulted from an acute toxemia, but, on the contrary, their outlines are sharply defined.

The vast majority of the axons and collaterals from the projection-cells show nothing abnormal. When the filaments are of extreme tenuity there are occasional breaks in the staining over short distances; then it recommences further on, and is then continuous over some short distance, only to be again broken. These variations of tingeing we consider due to an imperfect impregnation of the nerve-fiber, and not a pathologic condition. The terminal branches and the bulbous end-apparatus only stain infrequently, but wherever visible show the normal appearances by the phosphomolybdate method.

The bodies of the mossy neuroglia-cells are larger than normal, rounded, sometimes globular in outlines, and the tentacles are thickened and knotty. There are general evidences that these structures of the lymphatic system are undergoing modifications of a pathological nature. The support-neuroglia of various kinds shows no lesions.

Alterations in the cerebellum are upon the same order as in the cortex of the hemispheres, though not so well marked, the Purkinje cells, for instance, showing only occasional tumefactions of the branches and seldom any considerable loss of the gemmulæ. The nerve-fiber structures do not appear to be implicated in the lesions. It is apparent from the results of the last three studies that the cerebellar structures have a greater resistance in their cells to toxalbumins and toxins in general than the cerebral. Whether this is owing to a difference in the blood-supply, as has been suggested, is conjectural.

Discussion.—Dr. Francis X. Dercum said it was difficult—indeed, impossible—to estimate the wide significance of such researches, espe-

cially when we reflected upon the new trend observable in the minds of alienists in regard to the pathology of insanity. Certainly such well-known remarkable facts as that the toxicity of the urine in such diseases as melancholia was enormously increased, and the fact that in pneumonia the toxicity of the blood was increased, made it look as though the insanities themselves were due to changes in the cells owing to toxic substances circulating in the blood. It seemed as though we were approaching the day when that should cease to be a mere conjecture. Toxins had been discovered in other nervous affections—epilepsy, for instance. It had occurred to him that it might be well to make injections, not with ricin or some other well-known poison, but with some of those unknown, alkaloidal, basic poisons which were found in the urine of the insane.

Dr. Charles K. Mills said he did not feel that he was capable of discussing a paper of this kind, nor of doing more than merely expressing the great pleasure it had given him to hear it, and to congratulate the author upon the great work that he had done and was doing. As Dr. Dercum had said, it was certainly upon work of this kind that our real knowledge of the pathology of insanity and the relations of this pathology to symptomatology was to be based. It was certainly marvellous how, in a very brief time, the whole aspect of the question had been changed by the researches of Dr. Berkley and a few others to whom he had referred.

Dr. James Hendrie Lloyd did not feel like attempting to express a positive opinion upon this work except to say that it opened a vast new field, and one of great value, not only as to what it showed in reference to this particular series of experiments, but what it showed as to the possibilities of our future investigations of all forms of acute and, probably, chronic deteriorations of the cortex. He thought we were probably upon the verge of an entirely new line of work.

The President said the Pathological Society was indebted to Dr. Berkley for his kindness in giving the demonstration. It had been one of great pleasure and profit. *December* 12, 1895.

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2. *Abscess of cerebellum.*

Presented by M. H. Fussell, M.D.

S. K., aged thirty-eight years, Polander, speaks no English, and no definite history can be obtained. Never sick until two weeks ago, when

he was seized with an attack of follicular tonsillitis. I saw him in the attack; he apparently entirely recovered, and went to laboring-work for two weeks. He was then seized with severe frontal pain, which was worse on sitting up. Sight normal; no paralysis of any of the muscles.

July 10th. Deep inspirations with a tendency to sighing; no abnormality of chest or abdomen; pupils dilated but react to light; sleeps constantly and is difficult to arouse; pulse small and feeble. Complains constantly of head-pain, but there is not a solitary physical sign. He died comatose on July 20, 1895.

Postmortem, July 20, 1895. Body emaciated; rigor mortis not marked; wrists still warm, also back and abdomen.

Thoracic cavity. Lungs meet in median line; right lung slightly adherent to pericardium and to diaphragm; left lung slightly adherent posteriorly. Both crepitant and highly pigmented. Bronchial glands not enlarged; pericardium not distended, fluid normal in amount; heart normal in size; right side contains some liquid blood; left side normal; aortic and mitral valves perfectly normal. Right lung darkly pigmented on surface, but normal between lobes, also pigmented on section. Left lung in same condition throughout as right, but slightly adherent at back. Abdominal cavity contains no abnormal liquid; mesenteric glands slightly enlarged; appendix normal. Urinary bladder contains no liquid; small intestines normal. Spleen normal in size and appearance on section. Stomach: pyloric orifice easily admits index finger; the mucous membrane the seat of small hemorrhages; no signs of tumor; hepatic ducts are patulous. Liver normal in size; no growths or new formations. Left kidney tightly adherent apparently to spinal column, the adhesions having to be separated with a knife.

Right kidney not adherent and normal in shape and size; no cicatrices and normal on section. Urethra normal, also prostate gland. Bladder normal. Left kidney: connective tissue adherent to capsule, as it was to spinal column, requiring to be dissected off. Capsule markedly thickened in spots, but not adherent to kidney; tips of several pyramids whiter than normal and one contains a fibrous mass.

Brain. Dura mater is smooth and apparently distended, although the vessels are not markedly full. Just to right of median line is a spot the size of a silver quarter, the seat of a pachymeningitis; dura not thickened; the convolutions are flattened. On removing the brain a dram of stringy pus is seen between the cerebrum and the cerebellum. The ventricles are distended with liquid; the choroid plexus is pale. The right lobe of the cerebellum contains in the upper anterior portion two abscesses; there is a small abscess in middle lobe, and a fourth just

to the left of the median line. Indeed, the whole lesion may be regarded as a multilocular abscess. There were no cultures made of the pus, but it would seem that the abscess was due in all probability to infection from the tonsillitis. There was never any fever.

December 12, 1895.

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3. *Specimen from a case of trephining for persistent headache.*

Exhibited by R. H. Downes, M.D.

The woman from whom this button of bone was removed came under my observation on December 23d. I had seen her about three years before; complained of suffering from obstinate headache, and had been treated by many physicians, none of whom had ever found any history of head-injury. Upon examination I found a pronounced scar on the head, and obtained a distinct history of an injury several years ago, at the time of the receipt of which she was unconscious for fifteen minutes. The wound received no attention at the time. I decided there was a clot or something in the skull, and the history pointed to this. There was little loss of sensation on one side. The woman had never connected the headaches with the injury at all, but she recollected that her headaches commenced on the side that was injured. Upon pressure she had more severe headache. She had not gone one whole day in a week without some headache. I trephined and found a scar adherent to the skull, and the bone very thick. Under the scar fibrous tissue was forming. The dura mater was adherent at least one and one-half inches.

Discussion.—Dr. John B. Shober said he should like to congratulate Dr. Downes upon the result of his diagnosis. Of course, it was impossible to tell if the patient could be permanently benefited by the operation. The case interested him, as he had a somewhat similar one last year. A young woman had suffered severe fright and mental excitement at having seen her husband killed, and nine or ten months afterward developed epilepsy. At the time he saw her she was having attacks every week on Tuesdays at 5 P.M. She gave a very definite and clear history. It occurred to him that fright would not be the sole cause of epilepsy, and he inquired carefully into her case and found that nine years before she had been severely injured by falling and striking her frontal bone. There were a scar and a depression on the left side, and pressure over this scar caused severe pain. She complained of feeling

weak, saying that she had had two attacks that week, the first coming on the day after she had seen him, accompanied with severe headache. He had very little hope of doing any good by operation, but decided to trephine over the scar, and found a decided prominence of the internal table of the bone, which was distinctly pathologic. For eight weeks after the operation she had no attack, and then they reappeared as often as before. *January* 26, 1896.

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4. *Tumor of brain from a case of Jacksonian epilepsy.*

Exhibited by Joseph Sailer, M.D.

The specimen I show is from a person who suffered with Jacksonian epilepsy for the past year and a half, commencing in the left arm. Finally it became general with partial loss of consciousness. The patient subsequently developed paresis. The reflexes were uniformly diminished. There may have been some change before death. The lesion was a tumor growing in the dura mater, which projected into the brain-substance, and the convolutions were compressed. Microscopic examination will be made later. The brain shows this lesion in the motor center for the arm and in the ascending frontal and parietal convolutions on a level with the posterior portion of the second convolution. The tumor was examined microscopically and showed alveolar sarcoma.

February 13, 1896.

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5. *Hydrocephalus—internal and external.*

Exhibited by William G. Spiller, M.D.

The specimen is from an old man, Horace W., formerly a patient in the Philadelphia Hospital, who had been feeble for many years. For the past year he had not been able to walk. He had a peculiar chattering movement of the lower jaw. With the kind permission of Dr. Mills and Dr. Guitéras, I am able to show the central nervous system. At the autopsy the calvarium was found to be normal in thickness. The dura mater was extremely adherent, and in cutting this during the removal of the brain there was an escape of an enormous amount of fluid. After cutting through the corpus callosum the ventricles were found to be very large and the brain-substance much reduced in thickness.

On examination the corpus callosum is seen to be thin, and the gyrus fornicatus is much smaller than in a normal brain. The same may be said of the gyrus hippocampus and of the gyrus uncinatus, parts which are included in the rhinencephalon—to employ the term suggested by Turner—and which are supposed to be the portions of the brain in which the sense of smell is located, although the gyrus fornicatus is not believed by all writers to be associated with this function.

If the anterior commissure were of normal size, it would be a fact in favor of the congenital nature of the hydrocephalus, as in some of the lower animals it is present when the corpus callosum does not exist, and it would be interesting to note in the present case whether it were complementary to the corpus callosum. It is probably diminished from pressure. The gray commissure was probably absent during life. The gyri are very distinct; at the base of the brain there is some evidence of previous pressure, but this could not have been very great, and it would seem from this that it was a case of hydrocephalus due to cerebral aplasia—one of the so-called cases of hydrocephalus *ex vacuo*, where the fluid fills a space already existing—especially as the large amount of fluid external to the brain must have increased the pressure, and would have made the gyri less distinct if the brain had been originally of normal development.

The view of a congenital condition in this case is also strengthened by the fact that the rhinencephalon is proportionally smaller than the rest of the brain.

The preservation of the form of the brain indicates that the morbid change could not have occurred very early in the development.

In congenital defect of the corpus callosum the rhinencephalon is very imperfect, and the same is true in this brain, where the corpus callosum is very small. The explanation for this is not easy. The cingulum, a band of associated fibers from the frontal lobe to the inner and lower part of the temporal lobe, occupies a large part of the gyrus fornicatus, and its fibers are at right angles to those forming the corpus callosum. What, therefore, can be the connection in the hypoplasia of the corpus callosum and the rhinencephalon, for connection there certainly seems to be? Can we assume that collateral fibers are given off at right angles from the longitudinal ones which form the cingulum? If so, it does not seem probable that the number of these can be great enough to account for such a failure of development in the gyrus fornicatus.

The aqueduct of Sylvius and the fourth ventricle are much enlarged; but there is no hydromyelia of the cord as far as can be seen macroscopically.

Chiari, in Prague, has recently reported displacement and distortion of parts of the cerebellum, medulla oblongata, and pons in twenty-four cases of hydrocephalus, owing to the pressure in the cranial cavity, and in one case the cerebellum formed a spina bifida cervicalis.

All such changes have failed in this case, although the hydrocephalus was of high degree.

Discussion.—Dr. David Riesman said that in some of the cases there had been a history of an infectious disease in early life which caused the hydrocephalus, but the condition in this case was more likely to be congenital. There was an absence of granulations on the ependyma, it being perfectly smooth. *March* 12, 1896.

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6. *Some observations upon the lesions of the cortical nerve-cell in diphtheria.*

Presented by J. Dutton Steele, M.D.

For some weeks past Dr. Sailer and myself have been engaged in work in the Pepper Clinical Laboratory upon the central nervous system of a number of guinea-pigs that were killed by diphtheria-toxin, and that were very thoughtfully placed at our disposal by Dr. Joseph McFarland.

Our investigations have been pursued by the methods of Nissl and Golgi and the silver phosphomolybdate modification of Golgi's method by Berkley. Our work has been productive of certain results that we think best to put on record in this preliminary report.

I. I wish briefly to refer to the literature of the normal histology of the nerve-cell as studied by Golgi's method.

II. To the literature of its application to pathologic work.

III. To our observations by the Golgi and Nissl methods in our series of guinea-pigs killed by diphtheria-toxin.

I. *Normal Histology.* Modern writers describe the neuron of the pyramidal cells of the cerebral cortex as consisting of the cell body, branching protoplasmic processes usually called the dendrites, and an axis-cylinder, also called the axon.

The dendrites present at rare intervals, when studied by the methods of Golgi or Cajal, certain varicosities or tumefactions that occur in small

numbers and usually on the finer terminal branches of the dendron. A few writers mention fine lateral projections, described as spines (Fig. 1). Kölliker, in his edition of the *Handbook of Human Anatomy* for 1893, considers these varicosities and spines to be artifacts due to the uncertainty of the method and the possibility of the tissues not being fresh. Lenhossek, in the *Structure of the Nervous System*, edition of

FIG. 1.

Guinea-pig No. 4. Shrinkage and tumefaction of dendrites: loss of gemmules. Berkley's modification of the rapid Golgi method.

1895, admits the possibility of the varicosities being artifacts, but states that they are also found by the Nissl method, and inclines to the supposition that they are masses of chromophilic granules in the course of the dendrites. Stöhr, in his *Human Histology*, 1895, mentions the presence of small knots upon the dendrites, but does not comment upon them.

In the *Johns Hopkins Bulletin* for 1895 Berkley calls attention to the constancy of presence and true form of the fine spine-like lateral branches from the dendrites, which he calls gemmulæ. He inferred their physiologic importance from their constant disappearance in diseased conditions of the cortex.

II. *Pathologic Histology.* In the *Berliner klinische Wochenschrift* for 1894, Golgi reports certain observations made by him in 1874 upon the brain of a case of chorea, and other observations made at a later date upon cases of rabies occurring in man and the lower animals. He describes the presence of considerable numbers of varicosities upon the dendrites, beginning in the finer branches and gradually involving the whole of the neurodendron. This process was accompanied by atrophy of the cell itself. He considered these changes to be pathologic, because such varicosities could not be found in any such numbers in certain normal preparations, and because atrophy was present in the affected cells.

He concludes that the axis-cylinder has greater resistance than the protoplasmic processes or cell-substance. In one drawing he shows a considerable degree of tumefaction in the axis-cylinder of a Purkinje cell, but does not comment upon it.

In November, 1895, Berkley reported before this Society the occurrence of much tumefaction in the dendrites of certain rabbits poisoned by ricin, with the added observation that the gemmulæ seem to disappear in direct proportion to the advance of the process. In the winter number of *Brain*, 1895, Berkley reports findings in the brain of a series of rabbits poisoned by alcohol. He employs a modification of the methods of Golgi and Cajal—the silver phosphomolybdate method—for which he claims greater certainty of impregnation and finer definition of the dendrites and gemmulæ. He calls attention to the following divergence from the normal in the brains of the poisoned rabbits:

1. Direct shrinkage and diminution in size of the vast majority of the ganglion-cells of the cortex.

2. Certain swellings in the dendrites which commence in the finer processes. Some of the tumefactions are very small, some very large. These last excite the suspicion that they may be artifacts. Berkley, however, proves to his own satisfaction that they are not, by the demonstration of a regular striated structure by intense illumination.

3. The disappearance of the gemmulæ, especially upon the tumefactions.

4. Roughening of the stronger processes and to some extent of the cell-body.

He states that although he examined many axis-cylinders he discovered no change in them.

III. Our observations in the Pepper Laboratory thus far include four guinea-pigs killed by small doses of diphtheria-toxin. Two of the series, Nos. 1 and 4, died upon the fourth day from .008 gram per 100 grams body weight, the minimum fatal dose. Nos. 2 and 3 died in three days from .01 per 500 grams body-weight. The animals all died naturally, and the central nervous system was removed in from one to

FIG. 2.

Guinea-pig No. 2. Great shrinkage of the dendrites; gemmules almost gone. Rapid Golgi method.

four hours after death. They died from the effect of the toxins presumably, and showed no evidences of paralysis. Nos. 1, 2, and 3 were treated by the rapid Golgi-Cajal method, and No. 4 by the silver phosphomolybdate method of Berkley. A certain proportion of the pyramid-cells of the motor cortex show no change from the normal. The dendrites are not diminished in extent and show but few varicosities, and the gemmules are abundant and regular. Other cells appear to show abnormalities as follows:

1. A diminution in number and extent of the branching protoplasmic processes, giving one a distinct impression that the neurodendron is

shrunken. This is seen in attempting to use the camera lucida. One never fails to include all the dendrites of one of these affected cells in the field of No. 7 or No. 5 objective, while it is impossible to include the whole of a normal cell system in that magnification.

FIG. 3.

Guinea-pig No. 4. Shrinkage and loss of gemmules. Berkley's modification of Golgi's method.

2. A great increase in the number and size of the varicosities seen in the normal cell. In the cells least affected these are situated in the finer processes, and hence it may be supposed that the process commences in this part of the dendron. In the cells that are most affected they involve the larger or stronger dendrites. They are oval enlargements situated at irregular intervals upon the processes, and occasionally a very large arc is seen.

The cell-body and larger dendrites sometimes appear irregular in outline, as if they were eroded. This appearance strongly impresses me

with the opinion that such a condition of the cell is artificial, due to the brittleness produced in it by hardening in Müller's fluid.

Berkley, however, as I take it, considers this a pathologic change, although he calls attention to the tendency to brittleness just mentioned.

FIG. 4.

Guinea-pig No. 1. Moderate shrinkage and tumefaction of dendrites and loss of gemmules: tumefaction of axis-cylinder.

3. Loss in numbers and irregularity of the gemmules described by Berkley. This is very striking, whatever may be its significance. On the processes of neurodendrons that are not affected the gemmules are numerous and regular in distribution, but as soon as tumefactions

appear the gemmules diminish in number, apparently in direct proportion to the advance of the process. On the much shrunken and clubbed dendrites they are usually entirely absent, though occasionally one or two may be found upon the process near the tumefaction or upon its surface. In one preparation a process is shown and appears to be, as certain as such an assertion can be made to be, an axis-cylinder. This is shown in the illustration (Fig. 4). This process shows several distinct tumefactions, and appears to partake in the process affecting the dendrites of its cell.

I quote the following from Dr. Sailer concerning the findings by the Nissl method: "In the spinal cord the cells of the lateral column were stained a diffuse dark blue. The outline of the nucleus was lost, but the nucleolus persisted. Exactly the same changes were found in the large pyramidal cells of the motor region of the cortex. This change has been described by Nissl, who found it occurring after certain nerve-lesions produced for experimental purposes, and who regards it as a sign of advanced cellular degeneration."

The bloodvessels were for the most part unaltered. A very few were found in which it was possible to imagine a slight thickening of the intima and media, but nowhere could a perivascular round-cell infiltration be seen. Arteritis was, therefore, excluded. No hemorrhages into the tissue were observed, though carefully looked for. The neuroglia-cells were not well impregnated and so cannot be described.

Our observations agree in general with those of Berkley in the brains of animals poisoned by alcohol and ricin, and with those of Golgi in cases of chorea and rabies. It would seem to be the natural inference that the appearances described above, certainly pathologic in the finding by the Nissl stain, and surely a divergence from the usual appearance by the Golgi method, may be the result of the direct action on the ganglion-cells of toxins or poisons in general, and with no characteristic pathognomonic of any particular substance.

If this is true, and the above-mentioned tumefaction of the axis-cylinder is a correct observation, then this is a fact hitherto not definitely placed on record. It supplements Berkley's statement that the axis-cylinders in his specimens were not involved by showing that, though rarely the case, they can be affected by the degenerative process, and agrees with Golgi that the axis-cylinder resists degeneration longer, as a rule, than the rest of the neurodendron.

Discussion.—Dr. Joseph McFarland said it always seemed to him that when death occurred in diphtheria it must be on account of some

action of the toxin upon the nervous system. The postmortem appearances in death from small doses were generally so meagre that one could not tell to what to attribute death. It came on in a curious way, and depended upon the vital resistance of the animal as well as the amount of toxin. It would seem, however, as if the nervous influence played an important part. A horse in the course of their experiments had succumbed to a large dose without premonitory symptoms. The horse was suddenly stricken down as if in cardiac palsy, and the postmortem examination revealed nothing. Guinea-pigs usually died in from thirty to forty-eight hours, and when examined the postmortem findings were very slight. If the doses were minute, for example, like those mentioned by Dr. Steele, death might be protracted as long as ten days. After receiving the dose the animal seemed to suffer prostration, lost its appetite and weight, and the hair fell out, especially about the head. It usually died in a quiet position, generally with a straw in its mouth. The autopsy-appearances were less than in those that died from large doses. Small doses could, however, put the animal in the same condition occasionally. He remembered a horse that received 1 c.c. of the toxin and never recovered. He became progressively thinner and finally could not stand, dying from this one dose, which was simply given as an experiment to see how much he could stand. Sometimes 20 c.c. would not kill. In microscopic examination of the tissues there were found the areas which Drs. Abbott and Welch had described; but they would not account for the death. Alterations in the kidneys and other important organs were so slight that they offered very little explanation of the death.

He was very anxious to have Drs. Sailer and Steele examine these nervous systems to see if they could throw any light on the matter. He would like to ask Dr. Abbott if the little areas occurred from minute doses or from large ones.

Dr. A. C. Abbott said he could not trust his memory to say whether the doses were large or small. Dr. McFarland had credited him with the finding of the minute areas in the guinea-pig, but he had only called the attention of the Society to them. Drs. Welch and Flexner really had done the work, and Dr. Welch and himself had mentioned it. In single doses of diphtheria-toxin sufficient to destroy life, the areas of necrosis were always found. He was not inclined to think them as significant as Dr. McFarland did. They were spread through the body, not in every section he had cut, but in such organs as the heart, the muscles, the intestines, and the endothelial cells. They were very frequent in the spleen. He had no doubt that the changes Dr. Steele had shown

played an important rôle in the death of the animal, yet he thought that the necrosis found in the other organs had something to do with the disturbance of nutrition and the cachectic condition seen in the animals.

Dr. A. A. Eshner thought it would be very interesting to know to what degree these changes took place in animals that recovered, and so, if possible, to study the retrogressive stages that perhaps occurred in such animals, and to determine finally whether perfect recovery from the lesions observed in those cases ultimately took place.

Dr. David Riesman remarked that it was held by the majority of writers that the dendrites of one neuron were in contact, but not directly continuous, with the axis-cylinder of another, and that the transmission of nerve-impulses took place by contiguity. It had occurred to him that if the dendrites were lost, the transmission of nervous impulses would be difficult. If this should occur in the medulla oblongata or in some vital center where nerve-impulses took origin, if sufficient distance occurred between contiguous cells, the time might come when the impulses could no longer pass. The animal might then die, and the cause of death would be revealed by absence and shortening of the dendrites.

Dr. Steele said he had never seen anything in literature explaining the point raised by Dr. Eshner. He did not think any work had been done on it. *March* 12, 1896.

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7. *Multiple tuberculous tumors of the cerebral membranes and tuberculous deposit in the suprarenal bodies secondary to caries of the spine.*

Exhibited by F. A. Packard, M.D.

On February 10, 1893, there was admitted to the Episcopal Hospital a white male, aged thirty-six years, a native of Germany. On admission he complained of pain in the right foot and knee. His family-history was negative, as was also that of his past life. Twelve weeks before admission he began to complain of pain and jerking of the right leg. The toes became flexed during the jerking, and much interfered with walking. He also at the same time began to have headache, and noticed that his back became painful on rising from the stooping position. The pain gradually extended from the foot upward to the right hip. He was incapacitated from work for eight weeks before admission.

His health otherwise had been fairly good, although there was some loss in weight.

Examination on admission showed that he was thin, but not emaciated; that there was considerable lack of tone in the whole musculature; there was a marked prominence of the upper lumbar and lower dorsal vertebræ, and on walking the spine was kept very rigid. The knee-jerk was exaggerated on the right side, and there was ankle-clonus on the same side. A few days after admission he had a convulsion, beginning as a clonic spasm in the thumb and forefingers of the right side, which rapidly extended to the flexors of the forearm, then the arm, then the right side of the face, and ending with a general epileptiform attack involving both sides and all extremities. When the general convulsion began consciousness was lost for a short time. During the convulsion the tongue was severely bitten, but there was no incontinence of urine or feces. After this convulsion severe pain was complained of in the legs, but no convulsion until February 27th, when he complained of a nocturnal attack of cramp in the right arm and leg. This recurred on the night of March 1st and March 3d. On March 9th he had a general convulsion that began with twitching in the toes of the right leg. The same was repeated on March 13th. On March 19th and 20th he had attacks of nocturnal cramp in the right arm and leg, not followed by general convulsion or unconsciousness. Examination of the eye-grounds had been made, but with negative result, except for some overfulness of the retinal veins. Nothing of note transpired until April 29th, when he suddenly became delirious and required mechanical restraint. On the next day he was quieted by sedatives, but had to be kept under their influence for about six days. Even after that he had to have occasional doses of sulphonal, bromides, or other sedatives during the rest of the month of May. In June there was found some evidence of consolidation at the apices.

When I assumed charge of the ward, on July 1st, I dictated the following note: He is generally emaciated; the skin of the neck and face is dark and rather sepia in hue; on the surface of the abdomen the skin is exceedingly dark; on the face and forearms the dark skin shows a few scattered lighter areas looking almost like scar-tissue in their pearly whiteness. The pupils are equal and react well to light and accommodation. The face is symmetric, and there is no difference on the two sides on grimace. The chest is long and the intercostal spaces are very distinct; marked edema was present over the chest. The heart is found to be normal as to size and there are no murmurs, but the first sound at the apex is feeble; under both clavicles the note is rather high-

pitched, but elsewhere the percussion-note is rather hyperresonant; below the clavicles there is decided bronchovesicular breathing with increase of vocal resonance down to the second rib; over the rest of the left lung anteriorly there are creaking râles, with numerous subcrepitant râles on deep inspiration; on the right side the creaking sounds and subcrepitant râles are absent below the second rib; posteriorly the lungs are perfectly resonant, but at both bases the breath-sounds are decidedly "whiffing" in character. The liver and spleen appear to be normal. Examination of the abdomen shows marked tenderness on deep pressure. On the right side, two inches to the right of the umbilicus, there is a sense of deep resistance. The right knee-jerk is much increased, but no ankle-clonus can be developed. The muscular irritability of the lower extremities is much increased; plantar reflex is absent; there is no evident loss of sensation; the muscles of the legs and thighs are much wasted, but there is no absolute loss of power in any of the muscles.

Examination of the eye-grounds by Dr. Heyl on July 10th showed in O. D. evidence of descending neuritis with engorgement of the veins; in O. S. engorgement of the veins, but no sign of neuritis.

On July 11th he had a convulsion, beginning in the right foot, extending up the whole right side, and ending with a general convulsion with unconsciousness and great cyanosis. On the night of July 14th he had a slighter convulsion; he became gradually weaker. On July 24th he went into a stupor, and died on July 27th, without the development of any marked change after the making of the notes above recounted.

At the autopsy, eighteen hours after death, I made the following notes: Rigor mortis is absent; the body is much emaciated; the face, forearms, and lower zone of the abdomen pigmented. The skull is very thick; the dura mater free from adhesions to the calvarium; a large quantity of subarachnoid fluid is present; at one point in the parietal region of the left side the dura is tightly adherent to the brain-substance; on the under surface of the falx cerebelli there is an adherent, hard, yellowish-white mass of the size of a large pea, springing from the dura and readily removed from its bed in the cortex of the upper surface of the cerebellum; the arachnoid over the convexity on both sides is opaque and edematous; on the convexity of the left hemisphere at the point of dural adherence noted above there is quite a large growth of yellowish-white color, adherent superficially—*i. e.*, evidently springing from the dura. The centre of the area in which this tumor was embedded in the gray matter is one and three-quarter inches from the great longitudinal fissure; the tumor is irregularly oval in outline, its

superficial area being one-half by three quarter inches in diameter, its depth three quarter inches. An inch and a half below this growth is a pea-sized, hard, yellowish-white tumor attached to the pia-arachnoid.

On a line with these two tumors and bordering on the great longitudinal fissure is a growth of the size and shape of a medium-sized strawberry, attached to the dura by its base and readily rolled out of its bed of brain-tissue, to which it was not at all adherent. The growth first mentioned (the largest growth) is removed from its bed with some difficulty, tearing away a portion of the ascending parietal convolution; its bed is situated in front of the supramarginal gyrus and is formed by the ascending parietal convolution and the gyrus posterior to it. The smallest of the three growths rested in a bed formed at the expense of the supramarginal convolution. On the median surface of the left hemisphere is a small growth springing from the pia mater and having its bed in the convolution bordering upon the ventricle of the corpus callosum. It is quite circular in outline and measures 8 mm. in diameter. There are no superficial growths on the right side of the brain. The lateral ventricles are both slightly dilated, the left more so than the right. The optic thalamus of the right side looks larger than that of the left, possibly only apparently so from the larger size of the left ventricle. To the touch the basal nuclei are of normal consistence. The centrum ovale on each side appears to be normal, except for slight prominence of the puncta vasculosa. In the left optic thalamus at about its center (from before backward) is a hard nodular growth, readily shelled out of the brain-substance, of a yellowish tint, and measuring 7.5 mm. in diameter. In the lenticular nucleus of the same side, at the inner edge of the outer gray mass of the nucleus, is a similar growth, measuring 4 mm. in diameter. The caudate nucleus and internal capsule show no change. No growth is found in the right half of the cerebrum. The vessels and membranes of the base, the pons, and the medulla are normal.

On removing the skin over the spine the prominence of the lumbar vertebræ noticed during life is seen more distinctly. It is found to be due to a collection of white, friable, and, in places, cheesy matter mixed with bony detritus and a little pus. The finger finds the spinous and lateral processes and the bodies of the last two lumbar vertebræ and the posterior portion of the ilium very carious. The dura over the cauda equina is softened, and tears readily. A few strands of the cauda equina are distinctly softened and friable. The spinal cord itself shows nothing abnormal on inspection. In the upper lumbar region there are three small plates of cartilaginous appearance situated in the pia mater covering the posterior surface.

The organs of the abdomen are in normal position. The peritoneum is normal; the intestines of good color. There is marked enlargement of the mesenteric glands, and the lymphatic vessels of the mesentery are plainly visible as white cords; vermiform appendix thickened and bent upon itself. The pleura, pericardium, and heart are perfectly normal. The bronchial glands are enlarged and deeply pigmented, but there is no softening of their substance. Both lungs show at their apices some puckering with dense interstitial proliferation. On section both lungs are found stuffed with miliary tubercles at the apices, the distribution of them through the cut surface growing progressively less toward the lower lobes, so that at the bases only scattered groups of tubercles are to be seen and felt. The duodenum, small and large intestines are normal. The hepatic vessels and ducts are normal; the gall-bladder filled with thick, dark bile; the bile-duct patulous. In the notch of the liver is an enlarged, hard lymphatic gland lying near the hepatic duct. The stomach is normal, but in the serous coat near the cardiac orifice is a hard, round, white growth, measuring 1 cm. in diameter and 2 mm. in thickness. The liver is friable and evidently fatty; scattered through its substance are many whitish nodules, varying in diameter from 2 to 5 mm. The pancreas and spleen are free from growths. The semilunar ganglia are normal. The right adrenal presents itself as a large mass having the general shape of a cocked-hat; is firm, and on section shows a uniformily white and smooth surface; it measures 76 mm. in length, 51 mm. in height, and 19 mm. in breadth. There is no trace of normal suprarenal structure visible to the naked eye. The left adrenal is smaller than the right, but decidedly larger than normal; it measures 51 mm. in length, 25 mm. in height. In the medullary portion, at about the center, there is a focus of infiltration apparently similar to that present in the organ of the right side. In neither capsule is there any sign of cheesy change. Both kidneys are large, flabby, and congested. The vessels of the cortex are too plainly visible. Scattered through the cortical portions are a moderate number of whitish spots varying in size from mere specks to areas of 3 mm. in diameter. The prostate gland, seminal vesicles, bladder, and testicles are normal. Beneath the psoas muscle on the left side is a large collection of cheesy pus. In Scarpa's triangle on the right side there is a large abscess. The two lower lumbar vertebræ are very carious, as are also the upper half of the sacrum and the neighboring crest and wings of the ilia.

Microscopic examination of the growths in the cerebral membranes, of the left suprarenal capsule, and of various of the growths in the other organs, show the structure of tubercle and the presence of tubercle-bacilli.

The sequence of events in this case is rather hard to follow. Doubtless the old almost obsolete process at the apices of the lungs was the source of primary invasion. Whether the carious vertebræ caused the later and more widespread invasion of the organs and tissues and dissemination of the bacilli throughout the body cannot, of course, be positively stated, although taking the clinical history and the postmortem findings together such an explanation is borne out by all but one fact—*i. e.*, the gradually diminishing number of tubercles in the lungs from above downward. This distribution in the lungs would support the view that the pulmonary infection proceeded from the old apical trouble, and it is possible that the other deposits owned the same origin. The confinement of the cerebral growths to the left side is not explained by the findings at autopsy. *May* 14, 1896.

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8. *A case of noto-exencephalus.*

Exhibited by M. V. Ball, M.D.

Mrs. X. Y. Z., aged thirty-five years, the mother of five children, has had five miscarriages and is suspected of syphilis. The previous child was born very small and after a prolonged labor. Labor-pains came on Wednesday, May 13th, at 4 P.M. At 9 P.M. the os uteri was not dilated; at 6 A.M. Thursday it admitted two fingers. The pains occurred every fifteen minutes, but were short in duration. The abdomen on inspection looked no larger than that of a six-months' pregnancy, but the patient stated that in her previous pregnancies the abdomen was never larger, and felt sure that she had been pregnant the full nine months. The fetal heart-beats could be heard on the left side. The exact position of the child could not be determined by palpation. On digital examination a fleshy mass was found presenting, to one side of which, high up, a small ridge of bone could be felt. The mass was distinctly movable, and did not give to the touch the sense of a bag of waters. It was thought to be a misplaced placenta, although no bleeding occurred.

The pains continued all the afternoon, and, alhough the uterus was sufficiently dilated to admit four fingers, the nature of the mass or the kind of presentation could not be determined. Consultation was held with Dr. Modell, who was equally in doubt as to the nature of the tumor, and a third physician was called. Dr. Longaker, the last consultant,

diagnosticated " some sort of monstrosity," and the woman having been placed under chloroform the fetus was extracted without difficulty and without the use of instruments.

It was found to be a living female fetus, fourteen inches in length, presenting on the posterior portion of its head a huge tumor larger than the head itself. This tumor contained the cerebral hemispheres. The cranial cavity, owing to the absence of intracranial pressure, was very small and receding, the bones of the skull very small and firmly united.

The cerebrum was covered only by skin and membranes. The eyes stood out prominently, owing to retraction of the scalp. On the left hand a supernumerary thumb was well developed. Respiration and circulation were maintained for about fifteen minutes after birth.

The brain was very much congested and contained several clots in the ventricles. An examination of the internal organs has not been made. The monster belongs to the exencephalic group, and is, according to the classification of Hirst and Piersol, a *noto-exencephalus.*

May 14, 1896.

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9. *Brains from two cases of infantile cerebral paralysis.*

Exhibited by Frank White, M.D., and Wm. G. Spiller, M.D.

One of these brains is from a case of microcephaly, and it will be noticed that it is unusually small. The gyri and fissures, however, are well formed. The cord also is diminutive.

The other specimen is from a boy who had been hemiplegic. The entire area nourished by the Sylvian artery is sclerotic, and the cause, therefore, in all probability, is to be sought in an occlusion of this vessel.

The brains are merely presented as gross specimens, as both are to form the subject of microscopic study by one of us.

Discussion.—Dr. Rosenthal said it might be interesting to know the former history of this case of microcephalus of which Dr. Spiller had presented the brain. He had been treating the family for about ten years, and was present at the birth of the child. The child was referred by him to Dr. Dercum, who sent it to Dr. Keen, and it was among the first upon whom the linear operation was performed, without any result.

Another child in this family had hydrocephalus, and the third child began to have, about a week after birth, degenerative changes in the

mucous membrane of the mouth. This third child seemed to be the only one that had good cerebal functions. The father and mother were first cousins, and the mother was in constant fear of becoming pregnant and bringing forth microcephalic or hydrocephalic children.

Dr. Mills thought the presentation of such a case showed the inutility of anything like linear craniectomy in such cases. It emphasized the point that in 99 out of 100 of these cases it was the kernel as well as the shell that was arrested.

Dr. Laplace wished to amend the assertion of Dr. Mills by saying that a case of this kind showed the inutility of linear craniectomy in *certain* cases. *May* 28, 1896.

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10. *Case of ascending degeneration of the spinal cord following caries of lumbar vertebra.*

Presented by H. M. Fisher, M.D.

Henry F., a bricklayer by trade, at the age of twenty-five years fell from a building upon which he had been working, and was picked up unconscious. After regaining consciousness he complained of severe girdle-pains. It was not until four weeks later that posterior angular deformity was noticed. Five weeks later he returned to his work, the girdle-pains lasting for some time, but gradually disappearing. From 1876, eight years after the receipt of the injury, till 1886, the patient, from his own account, was as strong as he had ever been. At the latter date, however, he began to complain of getting easily tired in his legs and of dull, aching pains. Nine months later he found himself unable to walk without staggering. He, however, continued to work except at intervals, when he was at different hospitals for a few months at a time, until February 16, 1891, when he was admitted to the Orthopedic Hospital, leaving it November 10, 1891, and going thence to the Episcopal Hospital. At the latter hospital there was observed to be extreme rigidity of the muscles of the lower extremities—clasp-knife rigidity—marked increase of the patellar reflexes, and ankle-clonus. No impairment of the muscular power in the arms was observed.

Since last March there had been diminished power in the left hand, associated with some anesthesia, and much awkwardness in using this hand. Three or four months later the same conditions were observed in the right hand. Some wasting of the left interossei and of the left

adductor pollicis was observed; no marked wasting of the muscles of the lower extremities.

On resuming my service at the hospitals October 1st, I found that there were complete paraplegia and marked wasting of all the muscles; cough and dyspnea marked. Examination of the lungs anteriorly showed the presence of very numerous subcrepitant and submucous râles, but the patient was too weak to submit to a detailed examination. His dyspnea increased; he became rapidly weaker, and died November 25, 1895, a little over four years after his admission to the hospital and twenty-seven years after the receipt of the original injury.

The autopsy showed that the immediate cause of his death was a general tuberculosis, the lungs, spleen, kidneys, and peritoneum showing miliary granulations. Upon opening the spinal canal the body of the first lumbar vertebra was found to be distinctly carious, and the membranes over the body of this vertebra were markedly thickened and adherent. The cord, after removal and hardening in Müller's fluid, showed extensive sclerotic areas above the point of injury. One or two small hemorrhages had occurred into the substance of the cord above this point. At a point opposite the seat of the injury the cord showed almost complete degeneration—transverse myelitis. A little higher up the columns of Goll and of Burdach were seen to be chiefly involved. The lateral pyramidal and direct cerebellar columns were also more or less involved, but the degeneration was incomplete. In portions of the lower dorsal region almost no sclerosed areas were observed; higher up in the dorsal region the sclerosis was chiefly unilateral, involving the left lateral pyramidal tract. In the cervical region again there was symmetric bilateral sclerosis, involving the pyramidal tracts mainly. There were some areas of disseminated sclerosis also in the medulla. The pons, cerebral ganglia, cerebellum, and hemisphere were not submitted to a very careful examination, but were apparently free from disease. *May* 28, 1896.

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11. *Acute purulent meningitis.*

Presented by D. J. Milton Miller, M.D.

E. B., male, aged thirty-nine years, a machinist, was brought to the Episcopal Hospital in a delirious condition on March 29, 1896. The history given by his wife was that he had been well, except some head-

ache, until the day before his admission to the hospital, when he came home from work at 1 P.M. complaining of intense headache. A physician prescribed for him; but growing worse, and particularly on account of active delirium, he was removed to the hospital the next evening.

On admission the patient, a large, powerful man, was observed to have a discharging ear (left), and an inguinal hernia; the face was flushed; eyes injected; pupils contracted and regular; no retraction of head; no convulsions or history of such attacks. There had been some vomiting, but none after admission; the bowels were constipated. No swelling or tenderness in either mastoid region. Temperature 101° F.; pulse 82; respirations 38. The patient was violently delirious, constantly shouting and attempting to get up, necessitating confinement by straps and constant watching. Calomel was prescribed, ice ordered to the head, and bromids and chloral administered.

March 30, 1896. Patient became partially conscious this morning, and complained of intense pain in the frontal region. Bowels still constipated; temperature 102°; pulse 92; respiration 38. His wife says the ear has been discharging for six weeks. A quantity of urine has been obtained, an examination of which showed specific gravity 1024, 8 per cent. by volume of albumin, some pus-cells and granular epithelial casts. The temperature rose rapidly during the afternoon; the violent delirium returned, death occurring at 2 A.M.

Autopsy, six hours after death. Body that of a well-nourished adult; slight rigor mortis; no lividity of skin or edema. *Thorax.* Lung deeply pigmented, crepitant, and floats in water. *Heart.* Valves normal; atheromatous patches in aortic arch; chicken-fat clots in both cavities. *Spleen.* Normal. *Liver.* Normal. *Kidneys.* Right: Capsule somewhat adherent; substance somewhat swollen, otherwise appears normal; small cyst on surface. Left: Capsule easily stripped off; cortex and medulla seem normal; small abscess in pelvis. *Brain.* Dura not adherent; pia thickened and edematous; its meshes infiltrated with lymph and pus, which are particularly noticeable along the course of the vessels, where they are distributed in broad plates. This condition extends over the whole convexity, especially on left side. The lymph extends into the Sylvian fissures and over the cerebellum. Between arachnoid and brain there is about one dram of free pus; ventricles are distended with fluid and brain-substance is softened. There is no caries of the left petrous bone.

The extreme rapidity in which the fatal issue occurred in this patient is very interesting, death taking place in sixty-one hours after the acute onset. Noteworthy also is the amount of exudation in so short a period.

The cause was undoubtedly extension from the middle ear by means of the bloodvessels, as there was no evidence of necrosis, although the bone was not opened up. The kidneys were not examined microscopically, and the well-known difficulty in recognizing acute conditions in these organs from a macroscopic examination renders a positive statement as to their condition impossible. The appearances, however, were those of an acute parenchymatous nephritis. The very small abscess in the left pelvis is of interest. As there was no evidence of cystitis, it was probably part of the general infection, or possibly due to a small calculus. *May* 28, 1896.

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12. *Lesions of the brain found in a case of acute yellow atrophy of the liver.*

Presented by C. W. Burr, M.D., and Aloysius O. J. Kelly, M.D.

The clinical history of the case is as follows: B. D., male, aged forty years. His previous health had always been good. Having felt poorly for about one month, one week prior to his death he began to complain of chilliness, sweats, and vague gastro-intestinal symptoms. His wife then noticed that he was slightly jaundiced. The jaundice rapidly increased in intensity, until before his death it was extreme. On the second day of his illness he began to be delirious and to have general convulsions. The liver-dulness became very much decreased in area. The patient vomited often, and his bowels were absolutely constipated. The urine contained leucin and tyrosin, but no albumin. Two days before death his delirium passed into stupor, stupor into coma, and in coma he died, December 28, 1895.

The clinical diagnosis was acute yellow atrophy of the liver.

At the necropsy the liver was about one-third the normal size, brown in color, and with wrinkled capsule. It was peculiarly soft to the touch and cut like India rubber. On section the liver-structure was indistinctly visible in the right lobe, but absolutely abolished in the left. There was some oozing of bile. The gall-bladder contained about an ounce of thick, greenish-black bile, with a little bile-sand. There was no obstruction of the ducts, no calculi. The stomach contained a few ounces of bile-stained fluid, but was otherwise normal. The spleen was slightly enlarged and soft. The kidneys were congested; the capsule stripped well; the cortices were well marked off. The brain, macroscopically, appeared normal.

For the specimens we are much indebted to Dr. L. C. Peter.

The lesions discovered by microscopic examination of the brain may be divided into those found in sections prepared according to the Golgi method and its modifications, and those found in sections stained according to the Nissl methods and modifications. It is convenient to describe the latter first.

Sections from the motor and prefrontal regions of the cerebral cortex, after the usual procedures subsequent to hardening in alcohol, were stained with dahlia, vesuvin, magenta-red, methylene-blue, and thionin. While the majority of the stainings with the first-mentioned agents were very satisfactory, none of them approached in clearness and sharpness of detail those stained with thionin according to the method of Lenhossék. The stainings were far superior to those obtained with the much-vaunted methylene-blue, which latter were, however, better than those obtained with the other dyes. What follows, therefore, refers particularly to thionin-preparations, though precisely similar changes were disclosed by the other stainings.

FIG. 1.

Drawing illustrating parenchymatous degeneration of the cells of the cerebral cortex; implication of the nucleolus, nucleus, and cell-body. The cells in the upper half as in a microscopic field; in the lower half, collected from various regions. *N. C.*, normal cell for comparison. Thionin-preparation. × 1200. (ZEISS.)

The pathologic lesions discovered affect the nucleolus, the nucleus, and the cell-body (Fig. 1). Normally, the nucleolus is of a definite size, of rounded contour, with edges smooth and well-defined, and has a certain marked, not very variable affinity for the stain. In our prepara-

tions the nucleolus is very slightly, if at all, increased in size; it is of somewhat irregular contour, which irregularity is not marked, consisting when demonstrable of a slight and limited projection into the substance of the nucleus at one, occasionally two points, along its periphery; its edges, while sharp and well defined, are not as smooth as normally, and its receptivity of the stain is markedly increased, it appearing as a jet-black dot, rendering absolutely futile all attempts to recognize any structure. In its position within the nucleus we can recognize no deviation from the normal, it being situated here and there irregularly as normally.

The nucleus, while apparently exhibiting no marked deviations from the normal variation in size, is frequently distorted in shape, being often elongated, again quite irregular with projections and indentations along various parts of its periphery. The two or three larger and more deeply staining chromophilic particles normally present, irregularly arranged within the nuclei, are no longer visible as such. They have lost their identity by reason of the excessive staining which the other, finer, dust-like chromophilic particles have thus pathologically taken on. The normally clear karyoplasm evinces a decided affinity for the stain, being in many instances excessively stained. This staining of the normally clear karyoplasm, and the excessive staining of the nuclear chromophilic particles in many instances, produces such an intense staining of the nucleus as a whole as to obscure the nucleolus, which in other instances can be quite distinctly recognized.

The cell-body seems to be most intensely affected, there being present all gradations from slight implication to almost absolute destruction of it. The chromophilic particles, which normally are distributed throughout the cell-body with a certain regularity, usually in rows, are in our preparations very irregularly arranged, often showing very curious figures. We notice in some cells but a slight deviation from the normal, the chromophilic particles having disappeared from but a small area, and being possibly more densely aggregated in another region. From this slight change there are visible all gradations of destruction until there remains but the nucleus deeply stained, surrounded by a few particles still stained along the periphery of the cell-body. In many instances the chromophilic particles are excessively large. One is inclined to think of a coalescence of smaller ones, as high magnifications (1200) fail to reveal any distinction into parts. In cells manifesting all grades of destructive changes there is usually arranged around the nucleus a layer of fine, stained, dust-like particles, between which and the nucleus there is always a clear unstained zone.

In some instances the nucleus and cell-contents have been entirely destroyed, or the nucleus has dropped out of the cell, and all that is discernible are a few fine stained particles along what was the periphery of the cell. In many cells which are not so excessively affected by the destructive process, there is an appearance extremely suggestive of fat-globules and detritus. The fat therefrom may, unfortunately, have been removed during the process of hardening.

The protoplasmic processes as far as they are stained present in some instances changes similar to those described as affecting the cell-body. In most instances, however, they exhibit no deviation from the normal.

The pathologic changes described affect all varieties of cortical cells. We have been unable to discover that any one variety is more affected than another. In spots, however, the destructive process has been more marked than elsewhere. In certain regions all the cells in a quite considerable area are more or less destroyed, while in others but few cells are implicated. This applies to all the layers of cortical cells. Again, in a few places it seems as though the more superficial cells were more intensely affected than those of the deeper layers. These instances, however, are very few.

Other pieces of cortex after hardening in Müller's fluid were stained according to the silver phosphomolybdate method of Berkley. The difficulty of determining, by this and the ordinary Golgi method of staining, the number of cells affected is very great, owing to the uncertainty of the impregnation and to the fact that of all the cells of the various layers of the cortex but a very few are at any one time stained. This latter fact is, however, a great advantage, as we are thereby enabled to study much more readily the changes affecting the individual cells and their prolongations. This method of staining is inappropriate for detecting the finer changes in the cell-body, and in our preparations gave negative results. The changes affecting the prolongations are, however, quite definite. They are of two kinds, or, as we prefer to say, two stages of one process. The first consists of a swelling (Fig. 2), the second of an atrophy of the processes (Fig. 3).

The first are more numerous. They consist of irregular swellings or tumefactions which extend along the dendrites for a variable distance, and are of variable thickness. Some are very minute, others quite large. The apical process itself is very frequently affected close to the cell-body proper. In other instances the parent dendrite and its finer branches abruptly become thickened, and as abruptly resume their normal calibre. As a rule, throughout the course of these tumefactions the gemmulæ are absent, though occasionally one still detects a few projecting from the

FIG. 2.

Drawing illustrating irregular swellings or tumefactions of the dendrites, with loss of the gemmulæ, of a pyramidal cell of the cerebral cortex—early stage of degeneration. Golgi-Berkley preparation.

FIG. 3.

Drawing illustrating atrophy and disappearance of the dendrites—late stage of degeneration. Golgi-Berkley preparation.

periphery of the swellings. Along some of the finer dendrites not the subject of such pathologic tumefactions, the gemmulæ have also disappeared. In other instances they themselves appear somewhat swollen. The varicosities normally present at the point of branching of the dendrites appear in many instances to be excessively large.

The further stage of this destructive process has already been alluded to as an apparent atrophy. The gemmulæ have entirely disappeared from the dendrites—a very few may occasionally still persist. The dendrites themselves have become thinned and shortened. That they have not been broken or cut off during the process of preparation is evident from the fact that at their distal extremities they appear rounded and well defined. Further, many of the dendrites have entirely disappeared. In some of these latter instances, as well as in some of the former, the apical process is still quite thick.

This destructive process affects apparently indiscriminately the various cells of the several layers of the cerebral cortex. The determination of the number of the implicated cells is, however, as before stated, impossible. We could detect no pathologic changes in the axis-cylinders or collaterals.

The study of the cerebellum led to very indefinite results. Aside from variations in the intensity of the staining of the Purkinje cells, nothing of any note was discovered by the use of the aniline dyes. The impregnation of the cells of the cerebellum by the Berkley modification of the Golgi method was less satisfactory than was that of the cells of the cerebrum, but the alterations found in the Purkinje cells were practically the same as those described affecting the cells of the cerebral cortex. We detected no change in the neuroglia-elements of either the cerebrum or cerebellum, and no disease of the bloodvessel walls.

To sum up, we have found evidences of a marked destructive process affecting the bodies and the processes of the nerve-cells of the cerebral cortex. The lesion is parenchymatous, affecting the nerve-cells primarily, and is not secondary to any disease of the bloodvessels. We are entirely ignorant of the primary cause of this destructive process. It is more than probable, it is almost certain, that acute yellow atrophy of the liver is caused by a poison, and that it is not a local but a general constitutional disease—a toxemia. And since cerebral symptoms, delirium, perhaps mania, general or local convulsions, and coma, form no small part of its clinical history, we should expect to find distinct signs of disease in the cortex, whether such signs be the result of perverted function or its cause. The fact that heretofore microscopic study of the brain in conditions of delirium, acute mania, and the acute infectious

fevers has frequently been fruitless or indefinite, proves nothing, since we are only now beginning to learn methods of research competent to show any of the finer microscopic changes. As methods have improved, the number of so-called functional diseases has steadily decreased. Granting that acute yellow atrophy of the liver is a toxemia, we are confronted with the question as to whether the brain lesions found are due to the same poison or whether they are due to another or to others produced by disturbance of any of the body-functions. We cannot answer it. We have, of course, assumed that the lesions found by us in this one case are not accidental, but essential.

Whether this be so can only be proven by the study of other cases. The lesions found are, of course, not claimed to be characteristic of acute yellow atrophy of the liver. Indeed, we should expect to find similar changes in the acute infectious fevers accompanied by similar brain symptoms. As a matter of fact, the lesions found in this case by using Berkley's modification of Golgi's method correspond to those found experimentally by Berkley in ricin-poisning and alcohol-poisoning, and are not unlike those described by Andriezen and others as occurring in certain types of insanity, notably alcoholic dementia.

That the case was one of acute yellow atrophy of the liver is proved not only by the clinical history and the necropsy, but also by the microscopic examination of the liver, spleen, and kidneys.

Discussion.—Dr. Abbott considered this paper a very important contribution, for the reason that it threw a certain light upon the etiology of the affection. There were so many points dwelt upon by Dr. Kelly which were closely analogous in many respects to the conditions found in certain infectious processes that were characterized by toxic conditions, that it looked very much as if acute yellow atrophy of the liver, with its accompanying lesions and symptoms, might readily fall into that category. Of course, this was pure speculation, but it seemed likely that it was a toxic condition, probably arising from the intestinal tract. It was an interesting phase of that study of the day, the question of autointoxication. One of these preparations showed evidences of destructive processes in some of the nuclei, and he had no doubt if there had been more specimens we would have had more evidence of these local destructive processes going on.

Dr. Musser remarked that it was only further proof that acute yellow atrophy of the liver was not really a local disease, but a general affection. *May* 28, 1896.

13. *Senile paraplegia.*

Presented by Aloysius O. J. Kelly, M.D.

The clinical history of the case is as follows: G. B., aged seventy-nine years, widower, white, native of Scotland, jeweller by occupation, was admitted to the out-wards of the Philadelphia Hospital, May 3, 1895, and to the men's nervous wards of the same hospital, February 13, 1896, and the following notes obtained:

No history of any hereditary disease. He was treated in the surgical wards of the Philadelphia Hospital during September, 1895, for urethral stricture, and subsequently again transferred to the out-wards. He has been a moderate drinker; syphilis is denied. About three months prior to his admission to the nervous wards he noticed that he was weak in the legs. He never had any distinct apoplectiform attack. Two days before his admission, on attempting to arise in the morning he found that he was paralyzed from the waist down. He had retention of urine and incontinence of feces.

Examination on admission from the out-wards revealed complete paralysis of all the muscles below the waist. There were no areas of anesthesia. The third day after the paralysis came on, bed-sores began to form over the sacrum and heels. Although he was put on a water-bed, these bed-sores rapidly became worse. Examination of the urine showed a trace of albumin and a large number of granular casts. Symptoms of uremia began to develop, and spots of consolidation in both lungs could be detected. The fourth day after his admission he became delirious. The temperature never went above normal. He became rapidly weaker and died seven days after admission, apparently of uremia, February 20, 1896, 5 P.M.

Postmortem examination by Dr. Jamison, February 22, 1896, 11 A.M.

Body of well-nourished old man, a small bed-sore over the left heel and a large one over the sacrum. Bladder distended and inflamed. Kidneys showed parenchymatous and interstitial nephritis. Heart normal. Lungs emphysematous; a few spots of senile pneumonia. No gross lesions of the brain or spinal cord, except a chronic leptomeningitis.

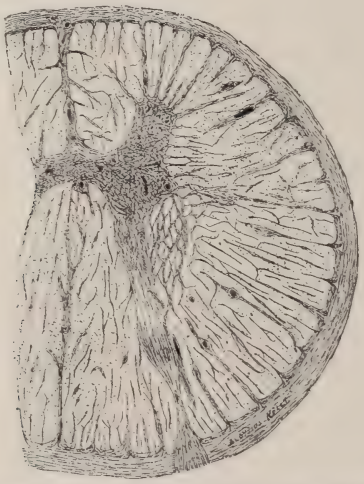
For the specimen (spinal cord) and the above notes I am very much indebted to Dr. Charles K. Mills.

Microscopic examination of the cord revealed changes which, though intimately associated in a causal relationship, it will be convenient to

describe under two headings, first, those affecting the nerve-fibers; second, those affecting other tissues.

Upper cervical region. In the posteromedian columns, close to and parallel with the posterior two-thirds of the median fissure, there is a narrow area in which the nerve-fibers are diminished in number and in thickness. There is a quite marked degeneration of cone-shaped areas with base inward, bounded by the anterior third of the posterior fissure, the posterior commissure, and the posterior cornua, and extending pointedly into the postero-external columns almost to the periphery. In the lateral columns not strictly localized to the pyramidal tracts, although there much more marked, the nerve-fibers are degenerated in part.

FIG. 1.



Drawing showing the excessive intraspinal overgrowth of connective tissue and its sources of origin, the sclerosis of bloodvessels and pia mater. Ammonia-carmin preparation.

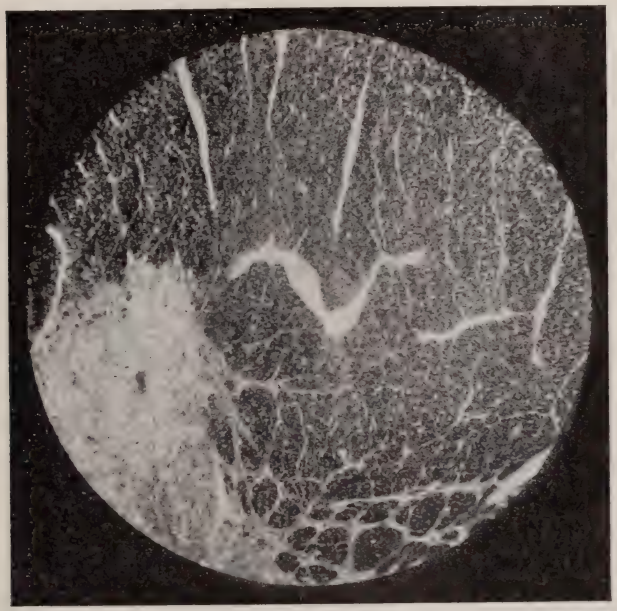
Cervical enlargement. The degeneration in the posteromedian columns along the posterior fissure is more marked than in the previous section, while the cone-shaped area alluded to is larger. The degeneration in the lateral columns is marked, but is not limited to the pyramidal tracts. While these tracts are much more affected than are other regions, the degeneration extends forward into the anterior ground-fibers.

Lower cervical. The degeneration in the posterior columns, while more marked along the posteromedian fissure, is distributed to some extent throughout the columns. The degeneration in the lateral columns, as heretofore, affects particularly and more markedly the pyramidal tracts, though it is not confined to them.

Upper, middle, and lower thoracic regions. The degeneration in the posterior columns is much more general and much more marked than in previously described sections. The degeneration in the lateral columns is of about the same extent as in the last-mentioned section.

Lumbar cord. The degeneration in the posterior columns is very marked. In the lateral columns as heretofore.

FIG. 2.



Photograph showing the bloodvessel-sclerosis, the associated perivascular connective-tissue overgrowth, and the numerically increased and much thickened trabeculæ of connective tissue and the numerous islets of sclerosis in connection therewith. Weigert-Pal preparation.

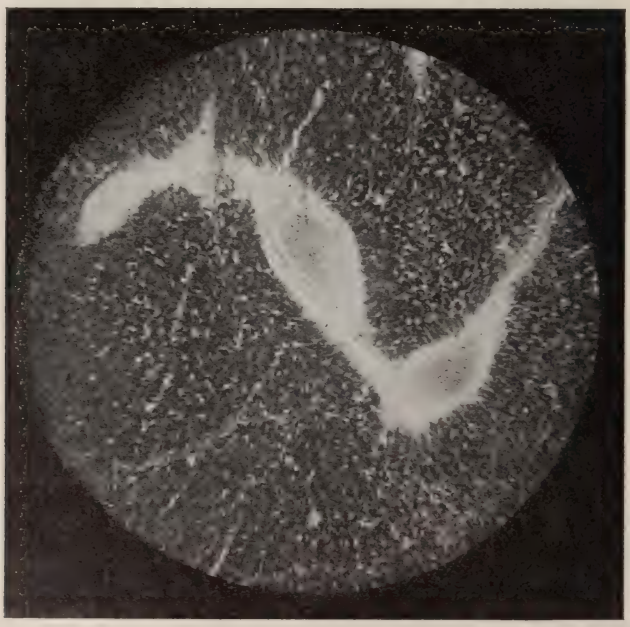
Sacral. The fibers along the lateral margin appear fewer in number and narrower than elsewhere.

When not otherwise stated, the nerve-fibers of the various tracts appear normal.

Throughout the cord at all levels, but especially in the regions noted as revealing nerve-fiber degeneration—that is, in the posterior and lateral columns—there is a great increase of the connective tissue. This connective-tissue overgrowth is intimately associated with changes in the bloodvessels and pia mater. With the exception of the very minutest ones, the bloodvessels are everywhere distended, and all have greatly

thickened walls. This applies to vessels of all calibers, from minute spinal ones to larger pial vessels. The smallest spinal vessels have greatly thickened walls, and the lumen of many of them has apparently become obliterated. Careful examination of somewhat larger ones reveals slight proliferation of the intima and great increase in the thickness of the media, which is poor in nuclei, concentrically laminated, and apparently somewhat hyaline in structure. Surrounding this latter is a layer of variable thickness of less homogeneously staining connective tissue. These changes are very marked in some of the medium-sized and larger vessels, which themselves are occasionally the seat of some round-cell infiltration.

FIG. 3.



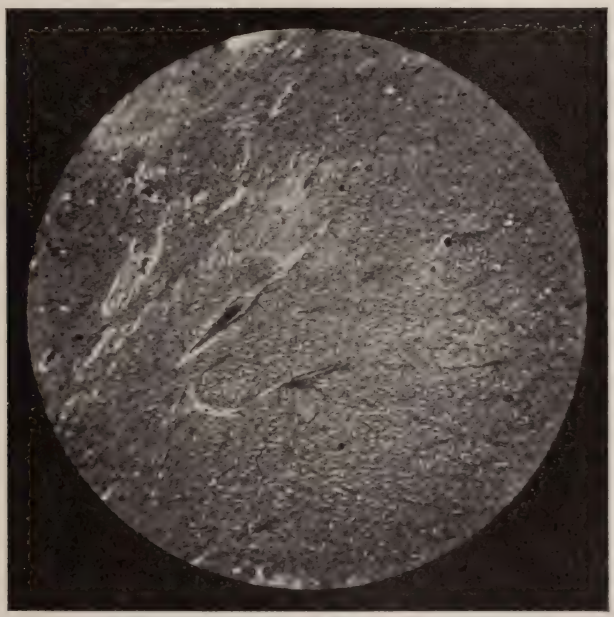
Higher magnification of Fig. 2.

The pia mater is very greatly thickened, contains very few nuclei, and has become united with the arachnoid. The peripheral layer of the spinal neuroglia is also greatly thickened. From this greatly thickened pia and neuroglia, and from the bloodvessels with thickened walls, as noted above, there extend throughout the cord numerous connective-tissue trabeculæ. These are very greatly in excess in number and much thicker than normally, and while present throughout the cord, are much

more in evidence in the areas already referred to as revealing nerve-fiber degeneration.

These trabeculæ are frequently very thick, inclosing within their confines bundles of nerve-fibers, whereas, again they are finer and surround only individual fibers, leading thus to degeneration of single fibers. The bloodvessel-sclerosis is, of course, not confined to the vessels of the white matter of the cord, but implicates of necessity those supplying the gray matter, which latter is not, however, appreciably altered. The central canal is filled with proliferated epithelium.

FIG. 4.



Photograph showing changes similar to those evident in Figs. 1, 2, and 3. Hematoxylin-picric-acid—acid-fuchsin preparation.

The most evident and important pathologic alteration discovered in these sections is, therefore, the excessive overgrowth of connective tissue; the nerve-fiber degeneration being very properly considered secondary thereto. The intimate association of this sclerosis with the bloodvessels is very patent. The bloodvessels themselves are the seat of arteritis and endarteritis, and around them as foci proceed trabeculæ of connective tissue encircling in places numbers, in others, fewer nerve-fibers. The neuroglia along the periphery of the cord is also greatly thick-

ened, and from it proceed also, into the cord, trabeculæ of fibrous tissue, much increased in thickness and in number.

This sclerosis originating around the vessels and periphery of the cord, while marked in all regions of the cord, is, however, much more intense in the posterior and lateral columns, especially in the area of the pyramidal tracts, and is more marked in the thoracic region than elsewhere. In areas slightly affected, more particularly where the sclerosing tissue surrounds groups of nerve-fibers, rather than individual ones, the fibers for the most part appear normal. In other regions, however, in which the sclerosing connective tissue envelops frequently individual fibers, these have suffered greatly therefrom and have become atrophied, axis-cylinder and medullary sheath in many instances having disappeared, in others being much narrower than normally. There remain, therefore, in consequence, many minute islets of fibrous tissue. In places where several larger vessels are in close apposition there are quite large spots of sclerosis.

There was discovered no embolic, thrombotic, hemorrhagic, or other process to account particularly for the acute manifestation of the severe symptoms. The case is simply an illustration of one of the many instances with which neuropathology abounds in which definite lesions are for a long time devoid of any very manifest symptoms. These, when they do come on, frequently make their appearance abruptly.

That the patient, however, was not entirely without symptoms prior to the acute attack of paraplegia, is evidenced by the weakness of his legs. And, in the light of the microscopic examination, and reasoning *a priori*, one may very safely assume that there were present other symptoms, unfortunately overlooked, as is but too frequently the case in the gradual and progressive weakness which attends old age. One cannot, therefore, but regret the imperfect history.

The designation "senile paraplegia" is admittedly ill-chosen, but has been selected because of its conciseness, and because it is sufficiently expressive of the clinical condition. We do not, however, desire to be understood as suggesting that the changes described are essentially senile in nature. They are distinctly pathologic, and, while frequently an accompaniment of old age, they are to be considered apart from changes of a purely senile character. It will hardly be appropriate here to enter upon the discussion of this subject in detail. For the photographs I am much indebted to Dr. Schively.

Discussion.—Dr. Charles K. Mills said that in the nervous wards of the Philadelphia Hospital there were at all times a large

number of senile patients. They were usually passed by as uninteresting cases; he had been long interested in them, although both the residents and the attendants gave them as little attention as possible. He had long wanted to know the pathologic conditions which were present, and he hoped this would be the beginning of considerable work in this direction. They arranged themselves into types, or at least they presented certain special features which separated them from other cases of paresis, if they be paretic. Sometimes there were cases whch seemed to be a combination of advancing slight paretic and ataxic disturbance, with general mental disability and general physical weakness. Of course, the very short shuffling gait, sometimes associated with tremor, and other phenomena, were present. Sometimes there were in these cases occurrences similar to that which took place in the instance which Dr. Kelly had reported, namely, that the patient, after having become step by step weaker, more paretic, more shuffling in gait, etc., would suddenly have an acute paraplegic attack, and might perhaps recover from that attack in a rather worse state than before, and go on, and later die of uremia, or it might be of another such attack.

Now, this case threw some light upon the chronic lesions which were present in these cases, and it also indicated a possible explanation of the acute attacks. In the wards at Blockley there was a large number of most interesting syphilitic spinal cases, which, among other phenomena, presented the history of an acute paraplegic attack similar to that which had been reported here to-night. One case of this kind was studied at the Polyclinic Hospital by Dr. Burr, and an account of it published in the *International Magazine*, with a lecture by himself, a year or two ago. There were syphilitic endarteritis and special involvement of the end-vessels; obliteration of vessels; some destructive processes, and extreme vascular disturbance; and then a clearing up. He believed that—not presuming that the present case was one of syphilitic disease of the vessels at all—in the senile case with changes such as described, there were processes in the cord, chronic and acute, similar to those which had been well studied and well described as occurring in syphilitic patients of earlier age.

Dr. Hutchinson (of Buffalo), present as guest, had been very much interested in the specimens of degeneration, or of senile paraplegia, from the fact that they seemed to him to illustrate the great drama which appeared to be going on in our bodies between the two great classes of embryonic tissues, the epiblastic and the mesoblastic cells. In the young, the mesoblastic tissues grew much faster than the epiblastic, in the intermediate states the balance being struck; whereas in later life

the mesoblast appeared either to undergo atrophy and to allow the epiblastic tissues to get the start of it, or else it became the seat of changes which pressed upon the more delicate epiblast and caused its destruction; the series of changes which took place in senile arteries, and the changes occurring especially in the nerve-tissues, and also in the secretory tissues of the body, appeared to be a sort of war between the epiblastic tissues on the one hand, and the mesoblastic tissues on the other. *May* 26, 1896.

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14. *The spinal-cord lesions in a case of fracture of thoracic and lumbar vertebræ.*

Presented by Aloysius O. J. Kelly, M.D.

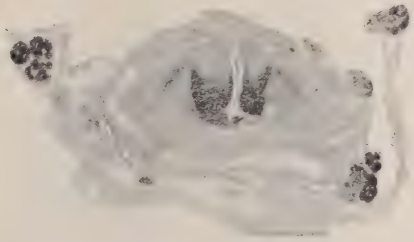
The clinical history of the case is as follows: W. T. A., male, aged thirty-three years, mason by occupation, was admitted to the Orthopedic Hospital under the charge of Dr. Wharton Sinkler, March 28, 1895. He had gonorrhea when he was twenty-five years of age, but syphilis was denied. December 30, 1891, a scaffold on which the patient was working gave way, causing him to fall twenty feet to the ground. He struck on his heels, then on his back. A brick standing on end struck him in the lumbar region to the right of the spinal column, causing a bruise which persisted for some time. On attempting to arise he could not move his legs. He began immediately to experience a sensation as of "pins and needles" from his waist down. For four days after the accident his legs were painful and felt cold. He had complete loss of control of his bladder and rectum. His sexual function was abolished.

Examination on admission to the hospital: No eruption or edema; deep bed-sores over the sacrum, on each heel, and on the calf of the left leg. No movements in the legs, no rigidity. Loss of control of the bladder and rectum. Complete anesthesia below the crests of the ilia; touch, pain, and temperature-sense all abolished. No girdle-pain. No tenderness on pressure over the nerve-trunks. Elbow-jerks equal on both sides; muscle-jerks in arms normal. Knee-jerks absent, not reinforcible. Achilles tendon jerks absent; plantar jerks absent; cremasteric jerks present, but much retarded. Abdominal reflexes present. Urine contains a trace of albumin.

Electric examination (Dr. Willets): No reaction to the faradic current in any of the muscles of the legs. Galv. Ancle. $<$ Kcle. 35

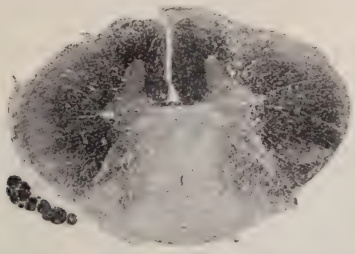
ma. required to secure reaction in tibialis anticus; calf muscles 30 ma. Galvanic qualitative and quantitative change. Pupils equal and react well to accommodation and to light.

FIG. 1.



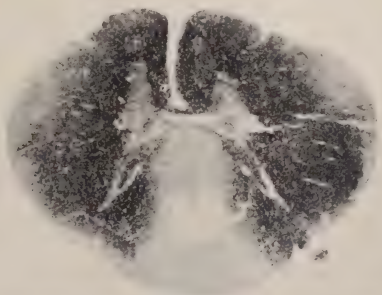
One cm. above lesion. Photograph showing distortion of the cord and complete degeneration of all the nerve-fibers except a few of those of the anterior column. Weigert-Pal preparation.

FIG. 2.



Thoracic cord, three cm. above lesion. Photograph showing degeneration of the nerve-fibers of the posterior column and of those along the anterclateral periphery (direct cerebellar and anterolateral ascending tracts). Weigert-Pal preparation.

FIG. 3.

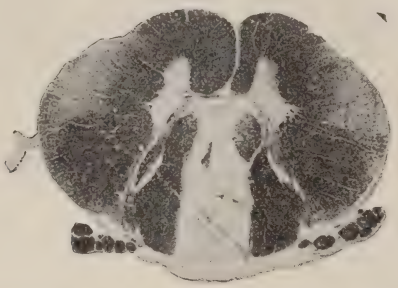


Thoracic cord, nine cm. above lesion. Photograph showing degeneration of the postero-median, postero-external (partial), direct cerebellar and anterolateral ascending tracts. Weigert-Pal preparation.

October 7, 1892, the patient died. The autopsy revealed a fracture-dislocation of the first lumbar on the last dorsal vertebra; the dura

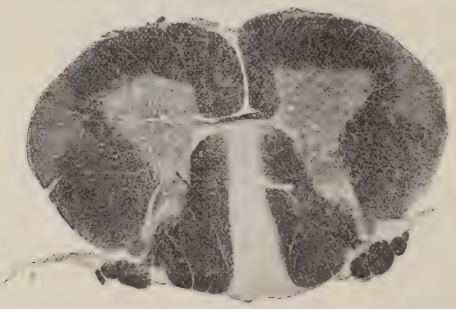
mater adherent to the bodies of the vertebræ at the seat of fracture; the entire lumbar cord and the lower dorsal cord much flattened; chronic cystitis; slight dilatation of the ureters; pyelitis; abscesses in the kidneys.

FIG. 4.



Lower cervical cord, sixteen cm. above lesion. Photograph showing degeneration of the posteromedian, direct cerebellar and anterolateral ascending tracts. Weigert-Pal preparation.

FIG. 5.



Cervical enlargement, twenty-two cm. above lesion. Photograph showing degeneration of the posteromedian, direct cerebellar and anterolateral ascending tracts. Weigert-Pal preparation.

For the specimen (the spinal cord) and the above notes, I am very much indebted to Dr. Charles W. Burr.

Microscopic examination (Weigert-Pal preparations): A section through the center of the area of disease reveals total destruction of the cord, it being impossible to recognize any of its various constituents. What was the cord is now a mass of fibrous connective tissue, through which run a few partially degenerated nerve-fibers. The nerve-fibers of the cauda equina, particularly the most external ones, still retain their myeline sheath. Surrounding the structures of this region is a thick mass of dense fibrous tissue.

A section through the cord, one centimeter above the former, shows

great distortion of the cord, particularly of the posterolateral regions, the component parts of which cannot be separately recognized. The cord is flattened from before backward, the posterior cornua being apparently displaced laterally, the posterior and lateral columns being crushed anteroposteriorly. The normal configuration of the anterior cornua and columns is well retained. There is complete degeneration of all the nerve-fibers in this region of the cord, with the exception of a few along either side of the anterior median fissure, a few of the most centrally located of the anterior columns, a few skirting the medial and anterior border of the anterior cornua, and a few of the anterior commissure (Fig. 1).

A section through the cord, 3 cm. above the first, shows the cord to be of about normal configuration. Along the anterolateral periphery there is a degeneration of the nerve-fibers of varying extent, being greatest just a little external to the posterior cornua. With the exception of a few fibers bordering the tips of the posterior cornua, the fibers of the posterior columns are degenerated in their entirety (Fig. 2).

A section through the cord, 9 cm. above the first, reveals a complete degeneration of the fibers of the posteromedian (Goll's) column, and some degeneration of the medial peripheral fibers of the posteroexternal (Burdach's) column. There is some degeneration of the fibers along the periphery of the cord between the anterior and posterior cornua of both sides. This degeneration is most marked about midway between the cornua, at which place it penetrates some distance into the substance of the cord (Fig. 3).

A section (lower cervical), 16 cm. above the first, shows complete degeneration of the posterior median columns, which degeneration toward the posterior commissure spreads out fan-shaped. The posteroexternal columns are otherwise unaffected. The degeneration along the lateral periphery noted in the previous section is more marked, and about midway between the anterior and posterior cornua extends quite a considerable distance into the substance of the cord (Fig. 4).

A section (cervical enlargement), 22 cm. above the first, reveals the same general characteristics as those evident in the last described section (Fig. 5).

Below the seat of manifest lesion—in the conus—it is absolutely impossible to recognize any spinal-cord structure. There is present a mass of connective tissue through which run a few partially degenerated nerve-fibers.

When not otherwise mentioned the tracts are normal.

There is, therefore, complete disorganization of the cord at the seat

of fracture, and above this region throughout the cord, ascending degeneration of the posteromedian, direct cerebellar, and anterolateral ascending (Gower's) tracts; and, for a short distance, degeneration of the postero-external columns. In other words, we find the anticipated spinal-cord lesions of a fracture in this region. To be mentioned only is the degeneration of the direct cerebellar tract in conjunction with a fracture of the vertebræ so low down.

For the photographs, I am very much indebted to Dr. Schively.

May 26, 1896.

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15. *A case of primary combined column-disease.*

Presented by JOHN H. W. RHEIN, M.D.

Mrs. L., aged fifty-nine years, presented herself for examination, complaining of unsteadiness in walking and numbness from the waist downward. Her family-history reveals a decided neurotic taint. Her grandmother and three aunts had paralysis agitans, a sister nystagmus, and her mother an attack of apoplexy. She herself had enjoyed excellent health prior to the onset of the present affection, which began, she stated, suddenly, about two years previously.

There was an attack of unconsciousness, preceded by stiffness in the neck and nausea. No paralysis followed, but shortly afterward distinct insecurity in walking, with progressive weakness in both legs, developed. She became emaciated and extremely pallid.

When examined by Dr. Frances Janney, eighteen months after the onset, she presented the following symptoms:

The patient was exceedingly anemic and moderately emaciated. There was no paralysis, though the legs were weak. The wasting was general, and did not suggest atrophy from central trouble. The station was poor, with feet together and eyes open or closed. The gait was distinctly ataxic. The knee-jerks were decreased on both sides, but reinforcible. The plantar reflex was retained. There was no change in sensation except paresthesia of the legs and trunk to the waist. A vaginal examination revealed the presence of a large tumor involving the fundus of the uterus. Its existence had hitherto not been suspected.

Some months later a second examination was made and the above condition confirmed. The knee-jerks were irregular. On the right the response was slightly below normal, on the left almost normal, on both sides reinforcible. There was no clonus or ankle-jerk. The

elbow-jerk and chin-jerk were present. Muscle-reaction was good. Pain-sense and sensation to touch and heat were normal. The paresthesia was still present.

There was no disturbance of the bladder or rectal functions. A fine rhythmic tremor in both hands was observed. This had existed for years and was not made worse by voluntary effort. An examination of the blood made by Dr. James E. Talley showed 2,464,000 red blood-cells and 40 per cent. hemoglobin. Dr. A. G. Thomson reported the eye condition as follows: Pupillary reaction normal, form-fields and fundi normal. Clear media, margins of disks hazy. The patient died a year later from exhaustion. Her exact nervous condition at the time was not studied.

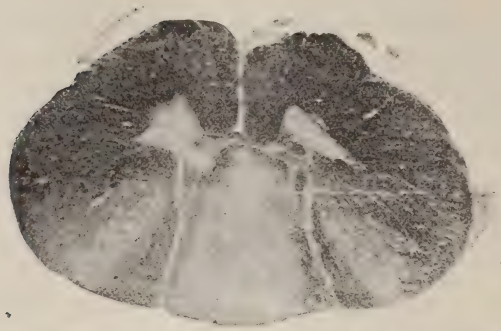
The necropsy revealed the following facts: In the uterus, involving the whole body of this organ, was a large, hard tumor, which microscopic section proved to be a fibrosarcoma. Some few masses, small in size and scattered, were seen in the liver, which was normal in size. The examination otherwise proved negative, except the spinal cord, which was the seat of a lesion presenting the greatest interest. Microscopically the cord appeared smaller than normal. There was no meningitis. The brain and its enveloping membranes showed no change. Unfortunately, the brain, in the process of hardening, underwent decomposition, hence a report of the microscopic condition of this organ is impossible. Macroscopically there was demonstrated degeneration in the posterior and lateral columns of the spinal cord, as seen by the distinct paling of these tracts. The process involved the whole extent of the cord and was most intense in the lower dorsal region. The degeneration in the lateral tracts was not so extensive as in the posterior columns at any of the levels.

The cord was hardened in Müller's fluid, embedded in celloidin, and stained by the Weigert-Pal method and with ammonia-carmin.

Microscopic. After staining, a sclerotic change was seen in the posterior and lateral pyramidal tracts throughout the whole extent of the cord, almost systemic in character. In the posterior columns the degeneration is most intense in the dorsal region, least in the lumbar region, while in the cervical region it is considerably more than moderate. There is always a strip of healthy tissue surrounding the degenerated in the posterior columns, and the columns of Goll are more intensely involved than the columns of Burdach.

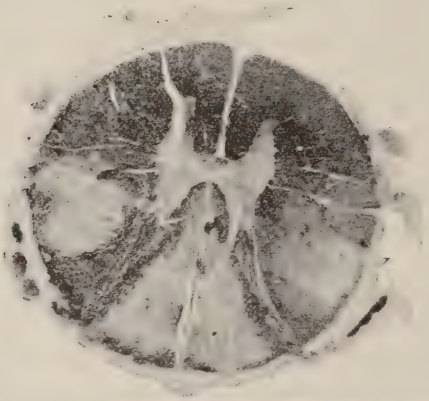
The ganglion-cells in the anterior horns show slight change in places, chiefly in the upper dorsal region. The posterior roots and Clark's column show no signs of involvement.

FIG. 1.



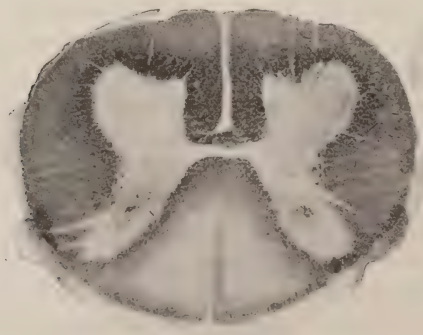
Cervical cord showing degeneration of the posteromedian, the postero-external (in part), and the crossed pyramidal tracts.

FIG. 2.



Thoracic cord showing degeneration in the posterior and lateral columns.

FIG. 3.



Lumbar cord showing degeneration in the posterior columns.

In the upper cervical region the degenerated area involves the columns of Goll in their entirety, except a few fibers medianward and cervically, which take the stain well. Though to a much less degree, the columns of Burdach are markedly involved. Centrally there remain a number of healthy fibers, while peripherally but a few well-stained fibers can be seen. The root-zones are present. In the lateral pyramidal tracts there is beginning degeneration, but to a much less degree than is seen in the posterior tracts. The anterior pyramidal and cerebellar tracts are intact. The anterior horns and posterior roots, as well as the remaining tracts of the cord, are normal.

In the cervical development the same condition exists save that the degeneration in the postero-external columns and in the lateral pyramidal tracts is more intense.

In the dorsal region the process has so far progressed that few healthy fibers remain in Goll's columns. In the columns of Burdach a narrow strip along the posterior horns alone is stained, the process growing more intense from above downward. In the anterior pyramidal tracts a very moderate degree of degeneration is observed. There is marked degeneration of the crossed pyramidal tract. In the upper levels a few ganglion-cells have lost their prolongation, but there seems to be no lessening in the number. The process involved to very slight degree the anterolateral ascending tracts.

In the lumbar region the degeneration is less marked, especially marginally. In the posterior columns healthy fibers in increasing numbers are observed. A band of well-stained tissue encircles completely the degenerated area.

In the lumbar enlargement only traces of the process in the lateral columns are seen. In the posterior columns are two wedge-shaped areas of degeneration on either side of the posterior fissure, surrounded by healthy tissue.

The degeneration now rapidly becomes less in extent and degree, until in the sacral cord there is no trace of the process visible. Clark's columns and the posterior roots are intact.

The carmin-specimens show smaller axis-cylinders; in places many entirely disappeared; a marked increase in connective tissue. To a very moderate degree is there increase in the bloodvessels. No granular cells were seen in any of the sections.

This is evidently one of a group of cases which has been variously described as combined tabes, posterolateral sclerosis, spastic tabes (Grasset), ataxoparaplegic tabes (Dejerine), ataxic paraplegia (Gowers), combined fascicular disease, progressive spastic ataxia (Dana)

true combined system-disease (Babinski and Charrin), and primary combined column-disease (Rothmann).

In 1871 Prevost described the postmortem findings in what was probably the first case belonging to this group. Later, Babesin, Strümpell, Siali, Westphal, and others added cases to the literature of the subject. In 1886 Grasset collected thirty-three cases with autopsies, and called the disease combined tabes (ataxo-spasmodique). The evidence did not point to a mere systemic lesion. He believed with Gowers that, while the lesion in the posterior columns was systemic, in the lateral columns it tended to be diffuse, and concluded that the process was a diffuse myelitis.

A study of the autopsies in the cases above demonstrates clearly that the disease is one with a most varied morbid anatomy, different columns in varying degrees of intensity being involved. The gray matter may or may not be diseased; the peripheral nerves have been found involved; Clark's columns and the posterior roots, as shown by Rothmann, Mayer, and others, are often affected. On the other hand, a number of cases show a noteworthy unanimity in the distribution of the lesion in the posterior columns. With moderate constancy the lesion is more intense in the lower dorsal region, less in the cervical region, and least in the lumbar and sacral regions. As a rule, there is a band of healthy fibers surrounding the diseased areas adjacent to the posterior horns. In the lateral columns the degeneration is not strictly systemic, as Gowers, Dana, and others have proved, and which is shown in the case under discussion.

In some cases the lateral mixed columns are encroached upon, and the direct cerebellar tracts are not infrequently involved. In Strümpell's case, reported in 1880, there was systemic degeneration of the pyramidal tracts, posterior columns, and cerebellar tracts, with atrophy of Clark's columns. The anterior horns and peripheral nerves escaped.

This variability in the morbid anatomy, together with the irregularity in the clinical history, suggests that the lesion is not truly systemic, but rather diffuse, beginning as a primary disease of the columns of the cord, the posterior and lateral pyramidal tracts most usually, and developing as the process progresses into a diffuse one. It is not a systemic lesion, as Strümpell, Hochhaus, and others maintained. It may, if the patient succumbs early, show a limitation to certain tracts in the cord, but when the disease is of long duration the process takes on an increasingly diffuse character. Strümpell believed that the further extension of the disease by continuity is improbable. Certainly, at first the limitation to certain columns and the freedom of the parts immediately adjoining the dis-

eased areas incline to this view, but later there is little doubt that continuity plays at least some part in the extension of the lesion.

The presence in the case under discussion of a malignant tumor of the uterus is interesting in connection with the recent reports of spinal degeneration in constitutions with morbid blood states. Lichtheim, Nonne, Minnick, Burr, and others have described characteristic lesions of the cord in pernicious anemia. Babes and Kalindero, in 1890, reported a case of Addison's disease, with lesions in the posterior and lateral columns with a distribution which suggests to no slight degree that found in many of these cases of combined sclerosis. Fleiner quotes Abegg's case of spinal degeneration in a case of Addison's disease. There was change in the posterior columns in two of his own cases.

Minnick mentions spinal change in three cases of chronic icterus, in one case of leukemia, in a case that died of tumor of the "vermis inf. cerebelli," in two cases of chronic nephritis, and finally, a case of carcinomatous cachexia with hydremia. Lastly, spinal lesion has been found in ergotism, pellagra, lathyrismus, and lead and alcohol-poisoning. This clearly demonstrates some relation between toxic blood conditions and spinal degeneration, but just what that relation is it is impossible in the present light of our knowledge to decide. In pernicious anemia the origin of the blood-state remains as obscure as the cause of the spinal lesion. The theory that the two conditions are common results of the same cause, probably a toxin, seems the most plausible explanation one is justified at present in making.

Since the exact duration of the tumor of the uterus is not known, we are inclined to place no importance upon its presence, and all the more likely to consider its presence a coincidence.

In studying the etiology of the affection it will be seen that nervous heredity plays an unimportant part; that syphilis is rarely an antecedent; that it is more frequent in males; that it is a disease of adult life; that exposure and excessive exercise predispose; and, lastly that it follows spinal injury, according to Rothmann. The symptoms are characteristic. The onset is gradual, the first symptom being unsteadiness in walking. Soon there is added stiffness in the muscles, though this may be absent.

Romberg's symptom is present and the gait is distinctly ataxic, though differing from the walk of true tabes. Thigh-raising and flopping down of the feet are not prominent. It is a mingling of the gaits of spastic paralysis and true tabes—when spastic symptoms are present. Lightning-pains are almost always absent, owing probably to the freedom of the root-zones. Other sensory symptoms are rare. The reflexes show a wide difference from the tabes. The knee-jerks are usually much exag-

gerated or spastic at first, while toward the last they may be almost or quite absent. The plantar reflex is present, as well as the remainder of the superficial reflexes. The sexual power is early lost. The eye-symptoms sometimes, but rather exceptionally, resemble those found in true tabes. The iris-reflex to light is usually preserved and optic atrophy is the exception. Progressive weakness ushers in the final symptoms.

The disease has little tendency in itself to cause death, according to Gowers and Dana, and usually is of long duration. Rothmann's experience, however, leads him to conclude that the duration of the disease is but three years. In our own case the patient expired just three years after the appearance of the first symptoms, but the case was not uncomplicated, and had the malignant tumor been absent we are warranted in supposing that the patient would have survived a longer period.

In conclusion, thanks are due Dr. Frances Janney for her kindness in permitting this report and for the opportunity of making the necropsy. To the kindness of Dr. Schively are we indebted for the photographs.

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Discussion.—Dr. Charles W. Burr said there was certainly one interesting thing about the specimen shown in this case. It was the fact that the changes were so much like those that had been described in instances where the anemia was great and of long standing. He had seen cases of anemia with spinal cords that presented exactly the same state of affairs. But he had not found in spinal cords which had come under his own observation, in cases of Bright's disease, or in cancer, or in sarcoma—no matter how great the anemia was—that the cord presented the same system-changes as in those of essential anemia. He thought that essential anemia, so called, was really a poison-disease, and that in sarcoma or cancer, whatever the poison might be, or whatever the cause of the condition might be, it was not a thing which affected the spinal cord the way it did in the essential anemia.

May 26, 1896.

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16. *Spinal cord from a case of Pott's disease.*

Presented by Henry D. Boyer, M.D.

The following case was under the care of Dr. J. P. Willits in the Germantown Hospital in the spring of 1894.

Unfortunately, he was an Italian who spoke no language other than his own, and his previous history could not be ascertained.

When brought to the hospital he was a middle-aged man, with the objective symptoms of a chronic myelitis. But no direct cause for the cord-disease could be found. He had evidently been in bed for some time. There were much emaciation, complete paraplegia, and contractures of the thighs and the legs, with fibrous anchylosis at the hips and the knees. The sensation was completely lost from the umbilicus down. The knee-jerks could not be tested on account of the contractures. There was full control over the bladder and the bowel-sphincters. The arms were in no way affected, except by the general weakness. The back was perfectly straight. There was no point of especial tenderness along the spine, nor any pain in any other part of the body. The temperature was at times hectic, but no focus of pus could be found.

This condition remained much the same for some five months, the symptoms of myelitis becoming gradually worse until death occurred. Toward the latter part of this time the control of the bladder and the bowels was lost, and a bed-sore developed on the sacrum and right heel.

The postmortem was made by Dr. Burr, who gave me the following notes of it and the spinal cord.

At the time of the man's death there remained the paralysis and the contractures of the leg, as before noted. There was no angularity of the spinal column and no prominence looking like an abscess near the surface. On cutting down on the spine there was opened a large pocket holding almost a pint of pus. This was found under and to the inner side of the right scapula. The pus had burrowed into the posterior mediastinum, but not into the pleural cavity. The laminæ of the dorsal vertebræ from the fifth down were carious. The bodies of the same vertebræ were markedly carious and filled with cheesy matter. The most marked destruction of bone was in the fifth, sixth, and seventh dorsal vertebræ. The meninges of the cervical portion of the cord were normal. Those of the lumbar region were almost free of deposit. In the dorsal region the dura was greatly thickened. At the point of most disease, from the fifth to the eighth dorsal vertebra, the cord was completely surrounded by a thickened dura, at some places one-half of an inch in thickness. The brain was normal. The lungs showed miliary tuberculosis with pleural adhesions.

The microscope shows the extreme dural thickness to be due to an inflammatory process in the dura itself, with new cell-formation, and to a cellular and a cheesy deposit on the outer side of the dura.

The cord is greatly pressed upon by this new tissue. It is distorted and nearly destroyed at this site by both the mechanical and some inflammatory processes. Secondary degenerative lesions are found both above and below this point. At the place of greatest pressure there are shown some inflammatory changes. Many new cells are present where only nervous tissue should exist.

There is no distinct line of demarcation between the gray and the white matter, both being almost totally destroyed. The cells in the anterior horns of the gray matter are few in number. Some are destroyed. Others are not perfect, having lost some of their processes. The central canal is filled by new epithelial cells. The white matter is mostly degenerated in all portions of the cord at this level. Most of the axis-cylinders and the myelin-sheaths are totally destroyed. Other myelin-sheaths are undergoing destruction, being swollen or narrowed, according to the degree of degeneration. Small particles of myelin, not in cylindric form, are everywhere found through the section. There are a few of both myelin-sheaths and axis-cylinders that are normal. These are found near the center of the section, and around the remains of the gray matter where least pressed upon.

The secondary degeneration both above and below the point of most pressure shows the same kind of change as above described, but to a less extent. It follows fixed columns. In none of these is the degeneration so marked as at the transverse myelitis.

In the cervical enlargement the posterior median tracts of Goll are almost totally destroyed. The columns of Burdach are a very little affected. There is another small degenerated area in the lateral ascending columns. This is close to the margin of the cord and in the extreme lateral part.

A section very high, near the medulla, shows the degeneration in the posterior median columns. That in the ascending lateral tract is not so wide as in the cervical enlargement. The other lateral tracts are normal above the lesion.

Below in the lower dorsal region all the lateral columns are involved, except the lateral fundamental zone lying near the anterior gray matter. The direct pyramidal tracts are also marked by the degeneration.

In the lumbar region the anterolateral tracts all show more or less destruction, except immediately around the anterior gray matter. The degeneration is greatest near the surface.

Below the lesion the posterior columns are not affected.

I wish to thank Drs. Burr and Willits for the privilege of presenting this case. *May* 26, 1896.

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17. *Cerebral syphilis.*[1]

Presented by Mary Alice Schively, M.D.

The case under examination presented the following clinical history: L. S., single, aged twenty-nine years; a seamstress by occupation. The family-history was negative. She had a history of alcoholism and syphilis, with sore-throat, skin-eruption, and loss of hair. In July, 1894, she began having persistent frontal headache; at the same time her eyesight began to fail. Upon awakening one morning early in September she noticed for the first time that her right eye turned upward and outward. Upon attempting to rise she was unable to stand, and discovered that her left leg was useless. She afterward regained some use of her leg, but continued to be subject to occasional attacks during which it would suddenly give way.

[1] For a short report of this case, exclusive of the results of the microscopic examination, consult Mills: "Some Phases of Syphilis of the Brain," Case V., Medical News, Dec. 7, 1895.

The patient was admitted to the nervous wards of the Philadelphia Hospital in December, 1894.

At this time she had a stupid expression of countenance, and wandered about aimlessly, speaking but little. She constantly complained of headache, which was relieved somewhat at intervals by repeated doses of potassium iodid. She had periods of marked irritability, which were followed in turn by mental hebetude, after which she would remain in bed and during which she could not easily be aroused, and would not answer questions. Her gait was of a shuffling character; knee-jerk was plus, and there was slight ankle-clonus of the left side.

Examination of the eyes was made by Dr. Oliver. The conditions were as follows: There was complete ptosis of the right eyelid; this eye itself was directed outward, and all movements of the globe were lost, except outward and a slight movement upward and outward; the pupil was immobile, 4 mm. in diameter. The left pupil responded to light. Both pupils were oval and both optic disks gray.

On February 28, 1895, the patient went to bed in a spastic condition similar to that of previous attacks, but more marked. The left side of the face was flushed and swollen, and she spoke only in monosyllables. During the night she had a convulsive attack followed by general muscular twitchings. After this the oculomotor paralysis became deepened, and she would not answer questions, although she appeared to understand what was said to her.

During the next day the mental dulness deepened gradually into coma. On the following day there was stiffness of the right arm, but relaxation of the left arm and both legs; knee-jerk was exaggerated on the left side. The left eye remained opened and fixed, the pupil being contracted. Later there was Cheyne-Stokes breathing. The patient remained in an apoplectic condition, dying on the evening of the second day.

The postmortem-examination revealed the following conditions: The dura was normal and not adherent; the pia free from inflammation, but much thickened; the meningeal vessels showed distinctly on the surface; the vessels over the hemisphere were distended. The right internal carotid was free until within 2 or 3 mm. of the penetration of the dura, and contained a flesh-colored, well-organized thrombus (Fig. 1). The right posterior communicating artery was a mere filament, while the left was much enlarged. The right anterior cerebral was very small; the left anterior cerebral very large. To the right side of the chiasm and the beginning of the optic nerve there was found a dense yellow mass surrounding the internal carotid and adherent to the oculomotor

and optic nerves; this mass fairly obliterated the Sylvian fissure in which it was lying. The left internal carotid contained a recently organized thrombus in the region from which the posterior communicating and

FIG. 1.

Drawing showing the gross lesions. *a*, right third nerve adherent to fibroid mass: *b*, right internal carotid plugged by a thrombus; *c*, fibroid mass filling the Sylvian fissure and extending backward to the crus; *d*, chiasm; *e*, left internal carotid containing recently organized thrombus; *f*, left third nerve; *g*, left postcommunicant at junction with postcerebral; *h*, basilar giving off postcerebral.[1]

anterior and middle cerebral form; this thrombus extended into the middle cerebral artery for about one-half its length. The cerebellum,

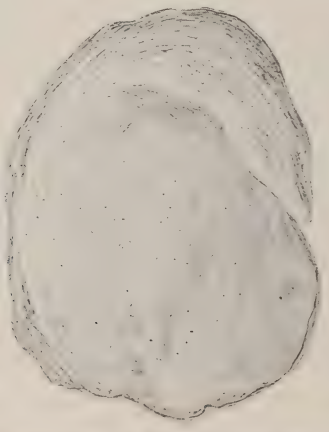
[1] This illustration is used through the courtesy of the publishers of the Medical News.

pons, and medulla showed on section nothing abnormal. Upon section of the right hemisphere, the head of the caudate nucleus, the anterior extremity of the striate body, and the lenticular nucleus were found to be softened. The material in this softened cavity was yellow and about the appearance of pus.

Through the kindness of Dr. Mills I obtained the above notes of the case from the Philadelphia Hospital Records, and also the specimen for microscopic study. The brain was hardened in Müller's fluid, and the sections were stained with hematoxylin and eosin; hematoxylin, picric acid, and fuchsin, and according to the Weigert-Pal method. As a result of the microscopic examination, I offer the following facts, illustrated in the accompanying drawings and photographs.

The oculomotor nerve of the left side shows in section the presence of scattered regions of small, round-celled infiltration, and some tendency to fibrous formation. The small, round-celled infiltration follows the course particularly of the endoneurium, separating the nerve tracts into smaller bundles, and that of the bloodvessels. The bloodvessels themselves are dilated; their walls thickened and surrounded frequently by groups of these small cells.

FIG. 2.

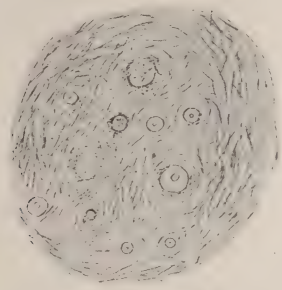


Section of right oculomotor nerve, showing complete degeneration and almost total disappearance of the nerve-fibers. Low magnification.

In one portion of the margin of the nerve nearest to the median line of the brain there is some evidence of degeneration of the nerve-fibers. Elsewhere the structure proper of the nerve is normal, the axis-cylinders being distinct.

The oculomotor nerve of the right side shows complete degeneration and almost total disappearance of the nerve-fibers (Figs. 2 and 3). Only

FIG. 3.



High magnification of Fig. 2.

the remnants of a few degenerated nerve-fibers are seen, the nerve-tissue being replaced by fibrous tissue, which is in turn in various stages

FIG. 4.

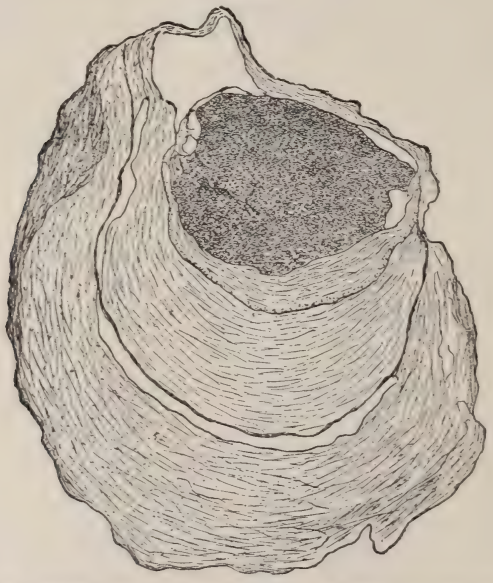
Section of right optic nerve, showing new fibrous formation, round-cell infiltration, and some degeneration of the nerve-fibers.

of caseation. The caseous remnants of a few minute bloodvessels can be detected. The position of the groups of partially degenerated nerve-

fibers is central and lateral. The epineurium is infiltrated with small, sphenoidal, densely staining cells, and this formation is continuous with the gummatous mass which surrounds the nerve. This gummatous structure consists partly of fibrous tissue which is very dense, and is infiltrated with small cells (similar to those seen in the epineurium), while other portions consist of more or less homogeneous substances without nuclei or cellular infiltration.

The left optic nerve exhibits changes similar to those affecting the left oculomotor. While there is present very little degeneration of the nerve-fibers, the round-celled infiltration and fibrous formation are more evident than in the other nerve. The epineurium, perineurium, and endoneurium, even to the finest divisions of the latter, are similarly affected. The round-celled infiltration follows also the minute arteries whose walls are thickened.

FIG. 5.



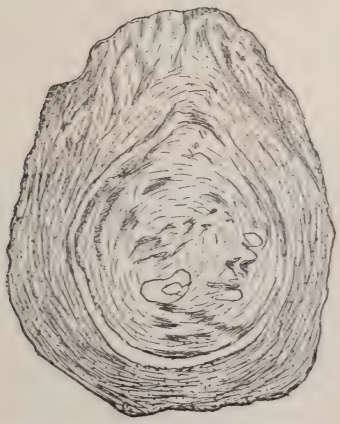
Section of the left internal carotid artery, showing a recently organized thrombus and arteritis proliferans nodosa.

The right optic nerve (Fig. 4) shows a greater extent and a more advanced stage of changes similar to those observed in the left. Particularly on the side of the nerve nearest to the gummatous mass (which invades this region of the brain) there is an interstitial infiltration of small spheroidal cells. In places these have progressed to spindle-shape

and fibrous formation. In some regions, especially on the side above referred to, there is in addition marked degeneration of the nerve-fibers. There are present also scattered areas of degeneration in neighboring regions, but none so distinctly marked as those above described.

The left internal carotid artery (Fig. 5) shows upon examination a recently organized thrombus which fills up the whole lumen of the vessel. There are present evidences of arteritis proliferans nodosa, the adventitia, muscular coat, and intima being much enlarged upon one side, while on the opposite side there is relative thinning of the walls, which are, however, thicker than normal. The adventitia is extensively infiltrated by small, spheroidal, deeply staining cells, which are here and there collected into dense masses. In some places there is a tendency toward caseation; this being shown by an indistinct staining of the nuclei; but the general tendency is not in this direction. The elastic lamina is intact. Evidences of obliterating endarteritis are shown by the increased thickness of the intima, due to increase in size and number of the endothelial cells in addition to the spheroidal cells.

FIG. 6.



Section of the right internal carotid artery, showing obliterating endarteritis, thrombus formation, and subsequent caseation of the entire structure.

In the right internal carotid artery (Fig. 6) are seen the conditions of obliterating endarteritis, thrombus formation, and subsequent caseation of the entire structure. The vessel-walls and the tissues in their immediate neighborhood are caseous for a considerable distance. The distinction of vessel-walls into adventitia, media, and intima cannot be

recognized, and there is no elastic lamina visible. In the periphery of the adventitia there is an invasion of small, deeply staining, spheroidal cells on that side of the artery which is nearest to the surrounding gummatous mass. This gummatous growth filling up the Sylvian fissure extends about half-way around the artery, and consists peripherally of dense granulation tissue; of small round cells with more centrally located fibrous tissue, infiltrated here and there with spindle and round cells; the center being a great mass of caseated tissue. The small cells are arranged in definite areas in some portions, but for the most part irregularly scattered.

FIG. 7.

Section of the right posterior communicating artery.

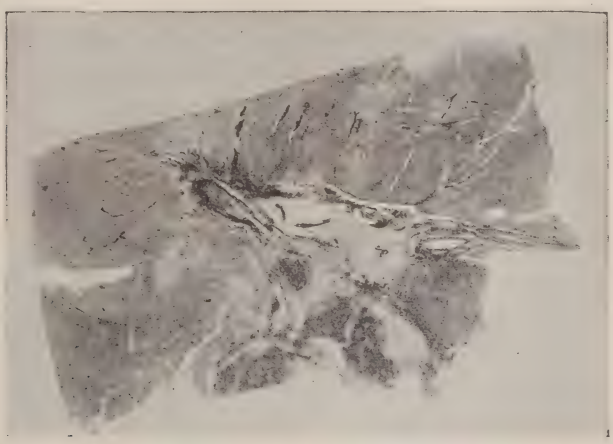
FIG. 8.

Section of the left posterior communicating artery.

The right (Fig. 7) and left (Fig. 8) posterior communicating arteries exhibit earlier stages, and minor degrees of changes similar to those affecting the carotid arteries.

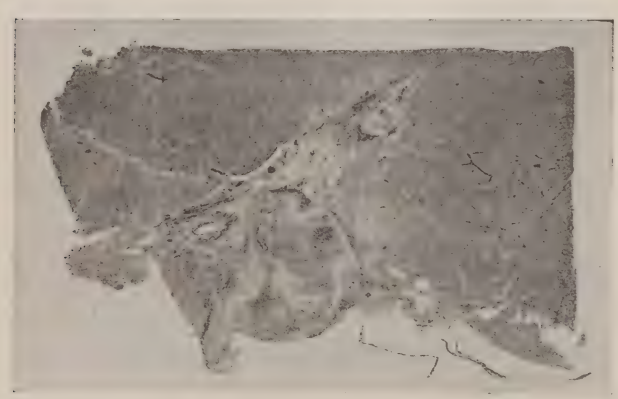
A section through the right Sylvian fissure and the gumma (Figs. 9 and 10) of that region scarcely permits of a distinction between brain-cortex, meninges, and vessels. Obliterating endarteritis and periarteritis

FIG. 9.



Section through the right Sylvian fissure, showing gummatous formation and caseation, endarteritis and periarteritis, and involvement of the meninges and brain substance.

FIG. 10.



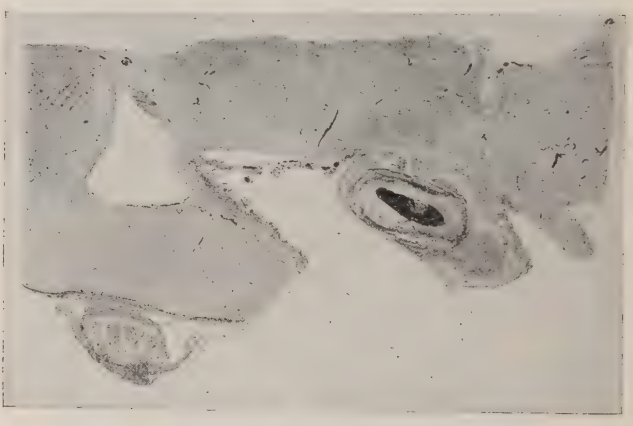
Section through the right Sylvian fissure, showing changes similar to those in Fig. 9.

have progressed in this region until the vessel and its thrombus form the center of a caseous gummatous mass, around which are regions of fibrous and granulation-tissue. There are numerous other foci of casea-

tion of lesser extent alike surrounded by fibrous tissue, spindle and small round-celled infiltration. The portion formerly meninges is now a layer of densely packed small round cells, and partly spindle-celled and fibrous or caseous. The infiltration extends a varying distance into the cerebral cortex in places, there being no area of limitation between cortex and meninges. The bloodvessels of the cortex are dilated, the walls thickened and surrounded by the same small round-celled infiltration.

A section through the left internal carotid and the optic chiasm (Fig. 11) reveals changes similar to, although less marked than those present upon the opposite side. The chiasm exhibits the already described dis-

FIG. 11.



Section through the left Sylvian fissure, left carotid artery, and optic chiasm, showing thrombus in left carotid artery and minor grades of other changes seen in Figs. 9 and 10.

ease of the support structures and some disease of the nerve elements. The implication of the bloodvessels is marked.

The pons Varolii shows evidences of gummatous formation, with caseation in the region of the pyramidal tract of the right side. The vessels of the pia mater in this region are enlarged and surrounded by spheroidal cells; the walls of these vessels are much thickened. There is some degeneration of the fibers of the pyramidal tract of the right side. In other respects the pons is normal.

In conclusion, the leading clinical features of the case may be summarized as follows: right oculomotor paralysis; paresis of the left leg; mental symptoms (aphasia, irritability, stupor) and a long apoplectiform period with convulsions preceding death. Pathologically, the case

illustrates most beautifully the multiplicity and diversity, both in nature and distribution, of the lesions of cerebral syphilis. The multiplicity of the lesions is evident in the various involvements of the blood-vessels, meninges, nerves, and brain-substance. The diversity in the nature of the lesions is manifest when we review in detail the arteritis proliferans nodosa, or, again, the arteritis obliterans simplex leading in places to multiple thromboses and localized softenings; the interstitial neuritis, with degeneration of the nerve-fibers, or the absolute syphilitic destruction of the nerve; the meningeal implication progressing in places to simple round-cell infiltration or fibrous transformation and fusion with the brain substance, in others to caseation; and the involvement of the minute vessels of the brain, which tissue is itself in places slightly implicated, again extensively so, as evidenced by softening, or loss of normal limitations and involvement in large gummatous formations, as in the pons and Sylvian fissure. There is hardly a manifestation of syphilis possible in the structures examined that does not here find its exemplification.

Discussion.—Dr. Charles K. Mills, in discussing the paper, said the case was most interesting, both clinically and pathologically, and he had little to say about it except that the admirable specimens and the excellent report commended themselves to the Society. Really, in the report upon this case, we had a report of almost every phase of syphilis of the brain, as presented either separately or in the same case: endarteritis, nodose arteritis, meningitis, cortical infiltration, isolated gumma, etc., throughout the entire list, and he thought the manner in which it had been worked up and presented was most admirable.

Dr. Spiller said the case described was another one of the interesting cases in which the attack came on acutely. Most of such cases were syphilis, and involved the cord as well as the brain. Indeed, some authorities, Dejerine, for example, said that 40 per cent. of spinal syphilis began with acute paraplegia. In the case that Dr. Schively had described paralysis in one leg came on overnight. In one of the cases which Sottas has reported death occurred after sixty hours, and the only lesions which could be found were of the blood-vessels. The explanation suggested was that the disease had existed for some time, probably, and owing to some over-exertion the spinal vessels were incapable of giving the necessary nutrition or performing their function, and suddenly ceased to act, and the cord was in that way rendered incapable of performing its functions. *May* 26, 1896.

18. *Meningocele; absence of one kidney.*

Exhibited by A. Hand, Jr., M.D.

This spinal cord is from a case of meningocele in the wards of the Children's Hospital, the clinical notes of which I do not know. Below the cervical enlargement the cord is seen to diminish abruptly in size and to consist of what feels like a strand of fibrous tissue. A microscopic examination, which will be made, will settle this point. The protrusion of the sac was in the lumbosacral region, the absence of the spinous processes beginning with the third lumbar vertebra. The only abnormality which this left kidney shows is compensatory hypertrophy, the right kidney and ureter being entirely absent, but the right suprarenal body being present. *September* 24, 1896.

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19. *Purulent cerebrospinal meningitis.*

Presented by M. H. Fussell, M.D.

Family-history: Father always enjoyed good health. At the age of forty-seven years he froze to death. His mother also enjoyed comparatively good health until the age of thirty-three years, when she died of smallpox. One brother was shot during a battle. He had one brother and one sister living; they are both in the best of health. He is married eight years, and is the father of two well-developed, healthy children; two children died in infancy. Patient is himself a fairly well-developed man, aged forty-seven years, and says he has never been sick, but has been a constant hard worker all his life. He is a German by birth, and has been in this country several years. He is on night-duty, loading engines with coal, in a place where he meets with a great deal of exposure, often having to work in the rain and in all conditions of weather.

February 4, 1897. Ambulance was sent to patient's home to bring him to hospital. The patient was found with his clothes on and walking about the house, though he seemed quite weak. Questioned about his sickness, he states that three days ago he began to feel bad; lost his appetite and felt as if he had taken cold. The following day he was unable to go on duty at night; he then went home and went to

bed, where he remained until the next morning before being brought to hospital. On admission his temperature was 101° F., respirations 28, and pulse 98, the latter of a very good character. The only thing he complains of is pain in the region of the left nipple; this pain is occasionally felt further around to the left side. The heart-sounds are normal. The lungs anteriorly seem in good condition; posteriorly at right base a few râles can be heard; otherwise they are in good condition. Liver and spleen normal. Urine examined and found to be normal. He expectorates very freely, but has no cough. Sputa examined and found to be free from pneumococci. On the third day patient's temperature reached 102.2° F.; he had three liquid stools, a result of a saline. His bowels previous to this time had been obstinately constipated. Within the following two days his temperature came down to 99° F., and finally to normal. For the next seven days (until twelfth day in hospital) his temperature ranged from 98° to 99.4° F. Patient seemed very much improved, and apparently was almost well. On the twelfth the temperature suddenly shot up to 103° at noon; he says he felt a little chilly during the morning, but did not have a chill. He complains of aching pains in the right side of his face and head, and slept very little during the previous night. On the thirteenth day his temperature was normal, and he appeared much better; fourteenth day, temperature 104° F. at noon. Blood examined for malarial organisms, but none found; during the afternoon he perspired very freely; at 6 P.M. he had a temperature of 103° F.; soon after this he became almost totally unconscious and developed typical Cheyne-Stokes respirations; he had not voided any urine for some time, hence was catheterized, urine examined again, and found to be normal. Cheyne-Stokes respirations continued until 8 A.M., fifteenth day. At noon temperature came to 101.6°; patient seemed brighter, but complained of some pain in his neck, especially when his head was moved. At 3 A.M., sixteenth day, temperature was 99° F., but by noon it reached 102.6°, and then by 9 A.M. the following day (seventeenth) it was down to 100.4°. Temperature began to rise again, however, and by 9 P.M. it had reached 104.6° F. He died one hour later. During the last forty-eight hours he did not void his urine, and it was necessary for him to be catheterized; he complained of some pain in abdomen; his neck seemed stiff, though there was no paralysis of the limbs. For twenty-four hours before death he was more or less delirious, but could until two or three hours before death be aroused, and would take nourishment and medicine. His pulse and respirations became extremely rapid for several hours before death.

While being catheterized it was found that he had a stricture about two and one-half inches from urethral orifice, and only a very small rubber catheter could be passed. There was no paralysis of the eye-muscles, and his eyes apparently were all right.

Postmortem, eighteen hours after death. No scars could be found on the body, which was very rigid. When the skin was cut through the superficial fat was found to be scanty. Superficial vessels bled quite freely. The bowel was very much distended; no fluid in abdominal cavity; thoracic cavity opened; organs in their normal position; lungs collapsed; no fluid in thoracic cavity; pericardium found to contain about normal quantity of fluid. Heart: right ventricle filled with jelly-clots and several chicken-fat clots; left ventricle contained jelly-clots and liquid blood; valves of heart normal; left lung normal; right lung had a few adhesions at apex, also at the base posteriorly; no consolidation. Liver normal. Kidneys: cortical substance in excess, capsule normal. White miliary nodules in right kidney; left kidney normal. Spleen perfectly normal. Mesenteric glands were not enlarged. The appendix was four inches long and lay inward and downward from the cecum; it was not thickened.

Brain. Dura mater not adherent. The longitudinal sinus contained a goose-fat clot. In removing dura from the brain the vessels of the pia mater were found to be filled with blood on each side of longitudinal fissure. On superior surface of cerebrum and on each side of fissure were two spots of purulent meningitis. Over the inferior surface of pons and medulla there was an adhesive purulent meningitis. Cerebrospinal fluid was in excess, and was not turbid.

Cord. Dura mater was not adherent, but entire arachnoid space was occupied by a very dense purulent exudation which was coextensive with the cord. Substance of the cord apparently not affected.

February 25, 1897.

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20. *A case of cerebral hemorrhage in a girl of nineteen, causing death.*

Presented by Joseph P. Tunis, M.D.

The history of the case is as follows: L. B., aged nineteen years, born in England. With her three-weeks' old child she was admitted to the Sheltering Arms, November 18, 1896. The heart and lungs were normal, her tongue clean, but flabby and tooth-notched. She was rather anemic and complained of frequent supra-orbital and occipital headaches.

On the evening of December 13th, while sitting at the supper-table and apparently well, she fell to the floor in a convulsion. When seen, a few moments after the attack, by the Resident Physician, Dr. Elizabeth M. Lovelace, she was able to answer questions. She denied previous similar attacks, premonitory symptoms, or aura. A specimen of urine was immediately obtained and analyzed; the reaction was acid, the specific gravity was 1015, and there was a half-inch band of albumin. Free catharsis was induced and a hot bath given. A slight headache followed the convulsion, but soon subsided, and the patient passed a comfortable night. A milk diet, hot baths, and Basham's mixture were prescribed, and after two days the patient was allowed to leave her bed.

The amount of urine secreted during the next three days was fifteen, fourteen, and sixteen ounces respectively. Repeated microscopic examination of the specimens discovered no casts. There was no edema at any time. The daily quantity of urine soon rose to forty and forty-eight ounces. The amount of albumin diminished, the headaches were less frequent and less severe, and the patient showed a marked improvement in every respect until the evening of December 18th, when she had a second convulsion. The posteclamptic coma lasted but a few minutes. During and succeeding the convulsion the patient's face pale and the temperature 98.4° F. A severe and obstinate throbbing with temporal and occipital headache of eight days' duration followed the attack. There was no increase of the amount of albumin nor diminution of the daily secretion at this time. She remained in bed twenty-eight days, during which period she became very hysteric, with an inclination to melancholia. On January 22, 1897, she seemed unusually well and cheerful during the day, but about 7.15 P.M., while sitting upon her bed talking with other inmates about her, she suddenly threw herself backward and became rigid. When Dr. Lovelace saw her there was neither convulsion nor coma; the irides reacted equally and freely to light; her temperature was 97.5° F.; her pulse 70, and the respiration irregular. Her face was cool and slightly pallid. Although she refused to answer questions, she seemed perfectly conscious. She was very restless and irritable, and at one time retched violently but ineffectually. As in a previous attack, a hydragogue cathartic and bromid were administered, and cold compresses applied to her head. At 9 P.M. she became quiet, and at this time the irides reacted well to light, the respirations were more regular and quieter and the pulse regular and steady; during intervals of extreme restlessness the pulse-rate had risen 10 to 12 beats per minute.

At 9.45 there was a marked change; the pulse was 60 to 63 and the tension increased, although not high; the respirations were slow, deep,

and stertorous; the right pupil was contracted to a pin's point, the left was two-thirds dilated; the irides were irresponsive to light, and the face very pale. She moved the left arm and leg when touched, but motion was lost on the right side. The lips were drawn evenly across the teeth and slightly separated, but there was no lateral distortion of the face. In a few minutes the right pupil was as widely dilated as the left; both angles of the mouth drooped and the cheeks puffed; both arms and legs were paralyzed; the pulse remained 59–60; the respirations assumed the characteristic Cheyne-Stokes rhythm; the coma rapidly deepened, and a few minutes before 11 P.M. the patient died, the heart beating a few times after respiration had ceased.

The last examination of the urine was made three days before death, when but a one-millimeter band of albumin was revealed. The specific gravity was never lower than 1015 nor higher than 1022. There was no evidence nor history of specific infection.

Postmortem. The body was that of a well-developed and perfectly nourished individual. There was no blanching of the skin and no evidence of anemia. Dr. Elliston J. Morris examined the abdominal cavity while I removed the calvarium. Everything in the abdominal cavity was apparently negative. There were no signs of peritonitis and no glandular or other evidence of syphilis. The kidneys were only examined after their removal from the body, and no microscopic sections were made of them. The capsule was not adherent, and there was no evidence of any decided change.

On removing the skull-cap the dura mater was unusually adherent and the Pacchionian bodies very distinct. The entire Sylvian fissure on the left side was filled with a currant-jelly clot, which followed the distribution of the branches of the middle cerebral artery. On removing the brain from the skull the fourth ventricle was found to be also filled with the same currant-jelly clot, and that seemed the most probable cause of her death. On making sections through the brain parallel to the corpus callosum the lateral and third ventricles were also found full of blood, the left lateral ventricle having much the larger quantity. I was unable to determine exactly the site of the original hemorrhage, but from the character of the clot it seemed most probable that the hemorrhage originated from two sources—externally from the middle cerebral artery on the left side, and internally from the arteries of the corpus striatum. There was no evidence of miliary aneurysms at the base of the brain.

The occurrence of cerebral hemorrhage in a woman aged nineteen years is so extremely rare that it has seemed advisable to put this case on record for that reason alone. *April* 8, 1897.

21. *A group of pathologic brains.*

Exhibited by WILLIAM G. SPILLER, M.D.

A few of these brains have been presented to this Society at previous meetings, and a few have been shown at other places, but a number of them are exhibited to-night for the first time. I must beg your indulgence for showing those which some of you may already have seen, but it is extremely difficult to form a collection of brains, each one showing a different lesion, and I am aware, also, that many persons now present have not viewed any of these specimens.

Cerebral abscess. The man from whom this brain was obtained had been hemiplegic, hemianopsic, and probably word-deaf. The case has been reported in detail by Dr. Mills and myself. The abscess-cavity in the left first temporal gyrus could easily have been emptied if it could have been diagnosticated. The patient had had bilateral papillitis, but had not had fever, nor disturbance of respiration nor of cardiac action. The most interesting facts in this case are: (1) The occurrence of epileptiform convulsions at the time of the first attack of unconsciousness, probably from irritation of the fibers of the internal capsule, this indicating that epileptiform convulsions do not always point to cortical lesions; (2) the location of the abscess and the ease with which it could have been emptied; and (3) the absence of degeneration in the external bundle of the cerebral peduncle after destruction of the first temporal gyrus.

Hydrocephalus. The brain which I now have to exhibit is a very good example of internal hydrocephalus of an intense degree. The layer of cerebral substance between the ventricles and the outer surface of the brain is much thinner than normal, and the callosum is so small that at first I thought it was absent. You will notice that the ventricles are enormously dilated. The patient from whom this brain was obtained was an old man with tumor of the lower jaw. His gait was feeble and stiff, and he was thought by some to be suffering from Parkinson's disease without tremor. According to Professor Marshall, the bulk of the corpus callosum, in proportion to the size of the brain, is twice as great in man as it is in the chimpanzee. "It is only fair to assume that the relatively enormous increase in the comparative bulk of the corpus callosum is due in large part either directly or indirectly to man's acquirement of language and all the mental and moral development that has grown out of it." (Bastian, *Lancet*, April 24, 1897.) I thank Drs. Mills and Guitéras for this specimen.

Microcephaly. The boy from whom this brain was taken was five feet three inches in height. The circumference of the head measured seventeen inches. The limbs were not in the least contractured. He was an epileptic and a low-grade imbecile; was helpless and unclean. The mother of this boy gave birth to feeble-minded twins. The father was a confirmed dipsomaniac.

The brain is one of considerable interest, and will be kept for more careful study. The size is much below normal, and the convolutions are most atypical in their arrangement. The pia over each hemisphere presented a shrunken appearance even in the fresh state.

Tumor of the vermis. I am unable to give you the clinical history of this case. The specimen has been shown by Dr. Lloyd before the Philadelphia Neurological Society. It is evidently a sarcoma, and is located within the vermis.

Cerebral paraplegia. This interesting brain was obtained from a child who had been paraplegic and idiotic. I would call your attention to the low type of development of the gyri; the first frontal fissure is almost as straight as though it had been marked with a ruler, and the first temporal fissure passes into the Sylvian. The first temporal gyrus is very short, and the posterior portion of it is reduced to a narrow strip. There are few giant-cells in the paracentral lobule, and the nerve-cells of the motor cortex are not as well formed as in normal brains. I thank Dr. Burr for this specimen.

Cyst of occipital lobe. We have in this brain from an epileptic patient, who died in status epilepticus, a most unusual condition of the occipital lobe. At first sight I thought it was a case of arrested development, but I managed to place my finger within the interior of the occipital lobe without damaging the brain, and was able to push out this contracted lobe to nearly normal extent. There appears to have been an old cyst in the occipital lobe. The cornu Ammonis is probably affected, for the mammillary body on the same side as the lesion is only about half the size of that on the normal side, and we know that the mammillary body receives fibers through the fornix from the cornu Ammonis. I regret that I have not been able to obtain any information in regard to the presence of hemianopsia during life. The child had had epileptic attacks since she was one week old. Her father had been burned to death seven months previous to her birth, and the mother was naturally much distressed. It seems difficult to regard this event as connected with the child's condition.

Spinal pachymeningitis. Although this specimen does not rightly belong to the group of brains exhibited, it nevertheless forms part of

the central nervous system, and is of interest. The child from whom it was removed suffered from Pott's disease. He had kyphosis of the cervicothoracic region. There was much pus within the lower part of the cervical canal external to the dura. The dura was adherent by its anterior portion to the bodies of the eighth cervical and first thoracic vertebræ, and was covered at this part by a cheesy, fibrous mass, which did not extend to the posterior aspect of the dura. The bodies of the lower cervical and first thoracic vertebræ were carious and yielded to slight pressure from an instrument. This boy suffered from intense dyspnea and probably died from paralysis of the respiratory muscles.

A limited lesion of the internal capsule. The following specimen has been presented by Dr. Daland. I show it again, as it is one of considerable interest. A small lesion, probably the remains of a small hemorrhage, has cut the fibers which, at a little lower level, form the knee of the internal capsule. The cicatrix is located in the middle portion, anteroposteriorly, of the tail of the caudate nucleus, and extends only a slight distance downward. The man suffered from dysphagia and dysphasia, but there was also a small aneurysm of one vertebral artery, which complicates the case.

Brain from a case of brachial edema in hemiplegia. I am indebted to Drs. Hare and Harris for the privilege of presenting this brain. Dr. Hare has reported the case as one of brachial edema occurring in hemiplegia. Before death, however, the lower limb of the same side was decidedly edematous, and at the autopsy the brain was found in the same condition. It was important to locate the lesion, as it might be possible in this way to find an explanation of the unusual degree of edema. A hemorrhage involving chiefly the external capsule, and probably due to rupture of the "artery of cerebral hemorrhage," was found at the autopsy. The location of the lesion, therefore, offered no explanation for the edema, and it was impossible to attribute the condition to destruction of the thalamus. The kidneys were very cirrhotic, and, if I may be permitted to express an opinion, I am inclined to believe that to this was chiefly due the excessive edema. In ordinary cases of hemiplegia edema is at times observed, and it may *in part* be the result of the muscular paralysis, for as the limb is no longer moved the circulation is impeded. We can easily notice this in permitting the hand to remain hanging by the side for a few minutes, when the veins become distended and enlarged. This loss of muscular movement is not sufficient, however, to explain the unusual degree of edema in Dr. Hare's case, and it seems to me probable that the condition of the kidneys was the chief cause. In a patient with probably diseased

peripheral vessels (for arteriosclerosis of some of the vessels was observed) and an abnormal condition of the blood, paralysis of the limbs might well cause edema of the affected parts.

Porencephaly. This brain presents a beautiful example of porencephaly. A cavity is situated at the lower part of the left central lobe and opens into the lateral ventricle. On the base of the brain, in the right hemisphere, there is another manifestation of hypoplasia, as the cortex is absent at this part. The left hemisphere is much smaller than the right. The convolutions radiate toward the crateriform cavity of the left central lobe, and this radiation has been considered by some writers as an indication of a congenital condition, but disregarded by others. The pia also extends into the depths of the cavity. The boy had not been paralyzed on the right side of the body, notwithstanding the fact that the fibers from the left motor region were entirely absent, and he had been able to walk everywhere, although with a limp. Neither had he been aphasic, though the fibers from Broca's region were certainly destroyed. The right pyramid, which contains the fibers from the normal hemisphere, was much larger than normal, and the left was entirely absent. It is probable that motor fibers passed from the right hemisphere to both sides of the body through the right pyramid.

Cortical sclerosis. A sclerotic area, about the size of a silver dollar and hard to the touch, was found in the right supramarginal and angular gyri of this brain from a boy who died in status epilepticus. A similar area was found in the right frontal lobe at the junction of the second with the third frontal convolution. The gyri in these two areas were very small as compared with the rest of the brain. On section the cortex in these sclerotic regions was found to be much thinner than normal, certainly not more than half the thickness elsewhere in the brain, and the white matter was also involved in the sclerosis. The microscope shows a great diminution of medullated fibers within these areas and an overgrowth of neuroglia.

The right hemisphere, probably as a result of these sclerotic foci, was five-eighths of an inch smaller anteroposteriorly than the left. No morbid changes macroscopically were found in the kidneys.

The boy had been a low-grade idiot during life. At the autopsy the right foot was found in the position of talipes equinovarus, and the left was said to have been in a similar position previous to a recent fracture. A point which seems to me worthy of special note is that the boy very frequently fell toward the right side—*i. e.*, toward the side of the sclerotic hemisphere.

It is not my purpose to go into details concerning these brains now to be presented to you, as Dr. Mills has already described them. I would only call your attention to a few facts.

In the brain of James Burk, a delusional lunatic, aged seventy years, who had attempted to commit suicide and had killed at least two persons, you will notice decided evidences of inferiority. The island of Reil is partially exposed on the right side, and there is almost complete confluence of the right central with the Sylvian fissure, and of the right calcarine with the hippocampal fissure; on the left side the confluence of the calcarine with the hippocampal fissure is only slightly less perfect. You will notice also that the left parallel fissure is connected with the Sylvian fissure. On the right side the parallel fissure is very long, and extends to the upper edge of the hemisphere. The type of fissuration of the frontal lobes is comparatively simple. The confluence of the central with the Sylvian fissure in this case is of interest on account of its rarity.

In the brain of Joseph Taylor, a murderer and a delusional monomaniac, one of the most interesting features is the appearance of the median surface of the right occipital lobe. The calcarine communicates in front imperfectly with the parieto-occipital, but completely with the collateral fissure. The occipital lobe appears to be cut off from the rest of the hemisphere on the median aspect. This and the preceding brain have been fully described by Dr. Mills in *Brain* for 1886, in his presidential address before the American Neurological Association.

Brain from a man, blind more than twenty-five years (*University Medical Magazine*, 1889). In this specimen the occipital lobes are very small. The details of the case have already been published.

Brain from a man, deaf-mute more than thirty years. The first temporal convolution of the left hemisphere is very narrow; in the right it is also somewhat smaller than normal.

These two specimens are of unusual interest as showing that atrophy of the cortex may occur, even in adults, after a long period when the functions of the part of the cortex in question have been destroyed. In the child arrest of cortical development after destruction of function is not so uncommon, but cortical atrophy in the adult is less frequently observed. I am inclined to place rather more value on the atrophy of the first temporal gyrus than on that of the occipital lobes.

Bilateral lesion of the upper temporal gyri. The most interesting brain I have kept until the last. It is a specimen which belongs to Dr. Mills and has been reported by him; but I am happy in being able to present to you the brain from a case of cortical deafness not

only for words, but also for sounds. The details will be found in the *University Medical Magazine* for November, 1891. The patient, fifteen years before her death, had an apoplectic attack which left her word-deaf, but not paralyzed. Prior to this first attack of apoplexy her hearing had been good, but after it she could not, by hearing, understand anything that was said to her. She could, however, hear music and sounds of various kinds. She could hear such sounds as a bell ringing and a clock ticking, until a second attack destroyed this power. She was not word-blind, for she could read the newspaper with intelligence. She was paraphasic, paralexic, and paragraphic. Nine years before her death she had another apoplexy, and deafness for sounds now became almost total. The superior temporal convolution in each hemisphere is greatly affected. We know that the posterior part of the left first temporal convolution contains the center for word-hearing, and Wernicke has taught us that word-deafness is due to destruction of the auditory center in the first left temporal convolution.

There have been four cases of double lesions in the upper temporal convolutions reported. They are :

1. The case of Kahler and Pick, 1879, in which there was loss of speech and hearing.

2. The case of Wernicke and Friedländer, 1883, in which the patient was completely deaf.

3. The case of Mills, 1891, in which the patient was deaf for words and later for sounds.

4. The case of Pick. This was a case of pure word-deafness, and yet there seems to have been considerable deafness to ordinary sound.

May 27, 1897.

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22. *Degenerative changes in nerve-cells.*

Presented by Joseph Sailer, M.D.

Of late years, since the introduction of the Nissl method, it has been possible to recognize pathologic changes in nerve-cells that were previously unknown. These changes have been studied particularly by Nissl himself, De Quervain, Pandi, Sarbo, Schaffer, Dehio, Marinesco, Centi ; and in this country by Stewart and Berkley. They apparently represent destructive processes going on in the cell itself and peculiar reactions to special morbific agencies. Pandi alone, of all the authors, has described reactions differing for each of the different poisons em-

ployed. The changes consist of alterations in the nucleolus so that it becomes irregular in outline, more eccentrically placed, usually at the wall of the nucleus, and develops vacuoles in the substance; alteration in the nucleus itself, so that, instead of refusing to take the stain as it does when treated by the basic aniline dyes, it becomes more or less diffusely stained; alteration in the chromophilic bodies, so that they either swell, become collected in masses in certain parts of the cell, or break up into fine granules; or, in some cases, can be observed to become smaller, less numerous, and this latter alteration can progress until they disappear entirely. Changes may also occur in the protoplasm of the cell. Normally, this stains very faintly by Nissl's method, showing with the highest power, apparently a delicate fibrous or granular constitution; it may, however, stain diffusely and more or less intensely in states of degeneration. The changes taking place in the chromophilic bodies bear a striking resemblance to those described by Klebs as occurring in degenerating nuclei of cells not belonging to the nervous system. In the nuclei of these cells the chromatin stains, as do the chromophilic bodies, with the basic aniline dyes. In the course of degenerative changes the chromatin may become increased in amount; it may be irregularly distributed; it may break up into masses or fine granules, or it may disappear entirely. These changes have been called hyperchromatosis, karyorrhexis, and karyolysis. No names have as yet been given to the changes occurring in the nerve-cells, but it may be possible to employ those before suggested by Klebs for the protoplasm of cells—*i. e.*, plasmorrhexis, plasmolysis, etc. The stage which corresponds to hyperchromatosis has already been called the pyknomorphous state by Nissl. He does not hold it to be strictly pathologic, having found it not infrequently in perfectly normal brains, but he admits it is much more common in the brains of animals that have been poisoned or in the neighborhood of focal lesions. I do not desire to draw any inferences from the similarity of the changes in the nucleus and the chromophilic bodies. It may be that the latter exert in the cells a function not unlike the function of the chromatin, and it is not unreasonable to suppose this because the nuclei of the nerve-cells are rather poor in chromatin. It is not, however, justifiable to suppose that they are portions of chromatin that have been included through the cells, for in certain of the lower animals, as I have been assured by Dr. Montgomery, they can be seen to develop in the protoplasm of the ganglion-cells. Nissl holds that they are not composed of nuclein, on what grounds I do not know. Cajal has lately discussed the subject, and applies the term chromatin to them, but leaves it in

doubt as to whether he considers them composed of nuclein or not. Benda looks upon them as simply filling up space between the reticulum, composed of the conducting substance of the nucleus. Nissl and Ramon y Cajal also look upon the unstained portion of the cell as a conducting structure, and the latter holds that the chromophilic bodies are simply reserved nutriment; but he seems to ascribe the same function to the chromatin of the nucleus.

I present this evening a section stained by Nissl's method taken from the medulla of a rabbit into whose brain a culture of anthrax had been injected and which died on the third day. It shows very beautifully the so-called pyknomorphous condition, the protoplasm and nucleus both taking the stain rather deeply, but not quite as deeply as the chromophilic bodies. The nucleoli in these cells also show irregularity of outline, displacement, and vacuolation. I also desire to exhibit some specimens obtained from the spinal cord of an idiot in whose brain gross lesions were found. The specimen has been stained by the Marchi method, and exhibits a degenerative change that was among the first recognized, that is to say, it consists of an excess of pigment in the nerve-cells. With osmic acid this pigment stained a deep black, although this does not necessarily prove that it is fatty in nature.

Discussion.—Dr. William G. Spiller said that a number of interesting papers had recently been written on nerve-cells stained by the method of Nissl, and four of them came at once to his mind. Biedl had just published a statement of the changes which occurred in the motor cell-body after section of the nerve-process. He found that the chromophilous elements underwent a granular change and finally disappeared.

Lenhossék had published a very thorough study of the spinal ganglia. His paper was too long for more than a brief reference. A clear zone was found at the outer limits of the cell; it varied in size in different cells, and this variation was probably the expression of activity or fatigue of the cell.

Dejerine and Sottas had recently examined the nerve-cells in a case of neuritis, and had been unable to find any alteration of the chromophilous elements.

Goldscheider and Flatau, in a series of careful experiments, had demonstrated that changes in the chromophilous elements did not always correspond to clinical symptoms, for they might be present without the latter. *June* 10, 1897.

23. *The giant-cells of the paracentral lobule.*

Presented by WILLIAM G. SPILLER, M.D.

At the last meeting of this Society Dr. Sailer presented some cells stained by the method of Nissl, and showing pathologic changes. I thought you might be interested in examining some of the giant-cells of the human paracentral lobule stained by the same method.

These cells are among the most interesting in the body. They are also among the largest of the human nerve-cells, with the exception of some of those of the spinal ganglia. They are only found in the motor area of the cortex, and are especially numerous in the paracentral lobule. They differ distinctly in form from the other cortical cells, and resemble very closely those of the anterior horns of the spinal cord. In type they are distinctly motor—*i. e.*, the chromophilous elements are rod-shaped and are arranged parallel to one another, and they differ greatly from the cells of the spinal ganglia, which we know are sensory. In the latter the chromophilous elements are granular and are not arranged in any distinct order in reference to one another.

I had intended to exhibit to you these giant-cells stained with toluidin-blue, which Lenhossék has recently recommended so highly; but my results have not been any better with this stain than with the thionin, which he formerly recommended.

Much doubt has arisen in regard to the nature of the chromophilous elements, but I believe the weight of opinion is in favor of the view that they are not coagulation-products. It would matter little for the study of pathologic changes if they were, for they are always of the same shape and arrangement in the same type of cells, and any alteration in them has been supposed to be pathologic. Many regard them as a kind of stored-up nourishment for the cell. In the giant-cells they offer a fruitful field for study in morbid processes of the motor cerebral cortex, for any alteration in the structure of these large cells is easily seen. *June* 24, 1897.

VII. THE DUCTLESS GLANDS.

1. *Tuberculosis of the suprarenal capsules, with pigmentary changes in the skin.*

Presented by J. D. Steele, M.D.

The specimens come from a patient in the surgical wards of the Philadelphia Hospital, who died from what seemed to be exhaustion due to a tuberculous arthritis of the knee and pulmonary phthisis. He had been confined to bed for some months, and, consequently, there was great prostration, but nothing to suggest the diagnosis of Addison's disease. There had been no vomiting.

Autopsy, performed at the Philadelphia Hospital, September 10, 1896. J. R., male, white, aged forty-eight years, a native of England. On the hands, over the fingers and knuckles, were areas in which there were decided changes in pigmentation. Some spots whiter than normal alternate with others in which the pigmentation appears to be increased. It is very difficult to say whether the pigment is increased in the darker areas or diminished in the lighter. Probably in neither is the amount of pigment normal. The areola of the nipple is markedly deepened in color. The pigment in the face is increased, but uniformly so, and evidently by sunburn. The man had black hair and dark eyes. There was no bronzing of the mucous membranes. There was recent pleurisy in the right side, with purulent fluid half filling the pleural sac. The right and left lung are both thickly studded with miliary tubercles which show no evidences of degeneration. In the lower part of the left lung there are several areas of caseous necrosis.

There is considerable fibroid change of the mitral and aortic valves. The myocardium is fatty, and there is some chronic interstitial myocarditis. The left adrenal shows two areas of thickening, 2 mm. in diameter, and of a grayish-yellow color. These are situated anteriorly in the upper portion, and do not show any degenerative change. The right adrenal contains a nodule 3 mm. in breadth and 15 mm. in length, occupying the central portion of the organ. It is of a bright yellow color, shows much caseous degeneration, and in two places is extensively calcified.

The cortex is thickened and congested, and has lost its characteristic color and appearance. About half of the organ appears normal. The semilunar ganglia and the larger nerves of the solar plexus appear to be normal both to the naked eye and microscopically. Microscopic examination of the adrenals gave typical pictures of tuberculosis; no tubercle-bacilli could be found, but the presence of epithelioid and giant-cells with extensive cheesy degeneration rendered the diagnosis easy.

The kidneys show a few scattered miliary tubercles. The liver is somewhat fatty. The thyroid gland is normal. The mesenteric glands are large and congested, and one has undergone extensive degeneration and calcareous infiltration.

There is a large circular ulcer in the ileum that has miliary tubercles on the peritoneal surfaces. There are similar but larger ulcers in the cecum, and five such in the sigmoid flexure, all tuberculous in character.

Discussion.—Dr. David Riesman said he had a suprarenal body from a case of Addison's disease. The gland was the seat of fibroid tuberculosis; the kidneys presented the lesions of acute Bright's disease, which might have been the result of some toxic substance in the blood and might have caused the patient's death. With reference to the rarity of pigmentation in cancer of the suprarenal, it might be explained on the same ground as the absence of glycosuria in some cases of cancer of the pancreas. In the case of the latter it had been assumed that the cancer-cells possessed the same secretory power, with respect to the internal secretion at least, as the cells from which they were derived. The same might be true of the suprarenal.

Dr. Joseph Sailer said in a case upon which he had performed an autopsy both suprarenal capsules were studded with minute miliary tubercles. It was an advanced case of tuberculosis. In that case a very careful search failed to reveal any pigmentation other than the normal increase in the hands and face and genitalia. There was no pigmentation of the mucous membrane. It was not common to have tuberculosis of the suprarenals without pigmentation.

Dr. A. E. Taylor said Dr. Sailer's case recalled one to his mind. The patient was a woman who had been sent to the University Hospital for treatment. It seemed to be a case of true Addison's disease. She was forty-eight years of age, and showed all the anemic symptoms. She was suffering from nervous disturbance incident to change of life, and menstruation ceased in six months. The pigmentation disappeared at once, although the case appeared at the outset to be typical Addison's disease.

Dr. Joseph McFarland recalled an article by Jacobi, in which it

was stated that the suprarenal bodies were for the peristaltic movement of the intestines.

Dr. J. D. Steele, in closing, said that in the series of cancer-cases recorded it was stated that in some of them the semilunar ganglion did not cause the pigmentation. The plexus had been deeply involved, but still no pigmentation took place. *September* 10, 1896.

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2. *Tuberculosis of adrenals.*

Exhibited by J. P. Arnold, M.D.

The specimens were obtained from the body of a woman who died in the Presbyterian Hospital. Mrs. A., aged fifty years, white, married; admitted August 26, and died September 1, 1896.

The patient complained of loss of appetite, emaciation, and progressive asthenia for the three months preceding admission. There were signs of advanced tuberculosis of both lungs. The heart-sounds were very weak, and the general weakness of the woman was very marked. There was some degree of tenderness in the epigastrium. The patient rapidly grew worse, and died in four days after admission.

The blood-count showed 3,960,000 red cells; tubercle-bacilli were present in the sputum.

Autopsy, six hours after death. Emaciated white woman. Post-mortem rigidity very slight. There was very marked brownish-yellow discoloration of the skin, especially marked on forearms, neck, and lower extremities. The middle line of the abdomen, the skin around the anus and genitals very dark. The glands surrounding the saphenous opening were enlarged on both sides. Advanced tuberculosis of both lungs, with numerous small cavities in both apices. The heart-muscle was very flabby, pale, and friable. The aortic and mitral leaflets were thickened, and there was atheroma of the aortic arch and of the left coronary artery. The right suprarenal contained eight or ten nodular masses, which were caseous in the center. The left suprarenal presented the same condition. Microscopic examination of these nodules showed the usual structure of the tubercle. No distinct bacilli were found. There was nothing about the region of the semilunar ganglia to attract especial attention. The spleen was large, weighing 240 grammes. The thymus gland was not persistent. *October* 22, 1896.

3. *Sarcoma of the thyroid gland.*

Exhibited by Alfred Stengel, M.D.

The following specimens, which I have the pleasure of showing by the courtesy of Dr. G. G. Davis, are from a case in the German Hospital. The patient, a man aged thirty years, presented no significant family-history or previous personal history. Three months before admission to the hospital a small swelling was noticed in the neck, and six weeks later this increased in size, at first rather rapidly, and then during the last three weeks very rapidly. There was little pain, but severe dyspnea, dysphagia, hoarseness, and cough. A distinct change of voice was subsequently noticed, and finally pains in the neck and radiating around the shoulders. On admission a rounded tumor, regular in outline, with apparent fluctuation, was discovered in the front of the neck. It was about the size of an orange, and more toward the left than the right. It moved in swallowing, though not freely. Operation was performed by Dr. Davis and nearly all of the growth removed. (Dr. Davis subsequently told me that he believed the whole was removed.) The growth was of a whitish color, firm, and encapsulated, of the size indicated. Within a few days a secondary nodule the size of a walnut appeared in the wound, and ten days after the operation this was quite large. A second operation was performed on the twenty-first day, revealing a large growth impossible to remove. The patient died on the thirtieth day.

Postmortem, a growth of the neck, extending from the sternum to the under surface of the tongue and anteroposteriorly from the vertebral column to the subcutaneous tissue, was removed. The cervical lymph-glands surrounding the thyroid were much involved in the neoplasm. The vessels were pushed aside and somewhat compressed. A number of secondary nodules were found in the lungs. No perforation of veins could be discovered, and the metastasis, therefore, most probably occurred in consequence of penetration of the lymphatics or of smaller venous branches. Microscopically the tumor proved to be a round-celled sarcoma.

This case is of interest mainly on account of the rapid growth after the primary operation. Rapidity of growth is not unusual in malignant growths, particularly after operations; but rarely do they increase in size so quickly as was here noted. *December* 10, 1896.

4. *Parasitic cyst of the spleen.*

Exhibited by S. M. Hamill, M.D.

This specimen is presented through the courtesy of Dr. John A. Johnston, Assistant Surgeon of the National Soldiers' Home, Virginia.

The man from whom it was removed was born in Ireland; he had been in this country since 1850. There was no clinical history accompanying. The report of the autopsy is as follows:

James D., aged seventy years; autopsy twelve hours after death.

General appearance. Body that of a very much-emaciated male; gangrenous spots over dorsum of left hand, with numerous similar spots on lateral surfaces of fingers of right hand.

Thorax. Muscular and adipose tissue of the chest very much diminished in amount; costal cartilages are not ossified; the pleura is adherent to the pericardium and to the chest-walls on both sides by delicate fibrous bands; the pleura itself is greatly thickened, and is of a deep pinkish hue.

Heart. The pericardium is thickened and the sac contains one-half ounce of straw-colored serum; the heart itself is small, pale in color, and very flabby; the right auricle is smaller in size than normal, and contains fluid blood; the tricuspid orifice admits the tips of two fingers; the valves are thickened, and the chordæ tendineæ are contracted; the right ventricle contains a large currant-jelly clot; the walls are thickened and show signs of fatty change; the left auricle is somewhat dilated and contains a chicken-fat clot; the mitral orifice admits the tips of two fingers, and the valve leaflets are atheromatous; the cavity of the left ventricle is small, and the walls measure one and one-quarter inches in thickness; the aortic orifice admits the tips of four fingers; and the valve leaflets are so atheromatous that they are brittle.

Lungs. The left was entirely hepatized; there was an area at the apex of the right lung about the size of an orange, that showed evidences of beginning consolidation. The other lobes were slightly emphysematous.

Liver. Enlarged, pale in color, and shows signs of fatty change.

Spleen. Smaller than normal; surface is corrugated and dotted with minute white spots. On section a yellowish hard mass was found lying exactly in the center of the organ.

Kidneys were normal, as were also the stomach, intestines, and bladder.

Sections made of the heart-muscle and liver confirmed the macroscopic appearance. The splenic mass was opened, and was filled with a whitish, cheesy mass. Cover-glass preparations made and stained after the Ziehl-Nielsen method were negative. There were no hooklets in the cyst contents.

There seems no doubt but that the specimen which is before you is a parasitic cyst of the spleen, and the two parasites which are most likely to give rise to cysts of this nature are the pentastomum denticulatum—the larval form of the pentastomum tænioides—and the echinococcus. According to Ziegler, the pentastomum is the commonest of the animal parasites infesting the spleen. It is, however, exceedingly rare in this country, but one case having been reported. It is not apt to occur singly; it frequently exists without injury to the individual, and when discovered postmortem it has usually been dead for some time. It then appears as a small pea-sized node made up of a mortar-like, chalky mass, enclosed in a fibrous capsule. Its hooklets may have disappeared. It will be observed that this description accords closely with that of the cyst under consideration.

Echinococcus-disease, of which this is more likely an instance, while it is quite rare in this country, occurs much more frequently than the pentastomum. Osler up to July, 1891, was able to collect 85 cases in this country and Canada. Sommers added 13 cases for the United States in 1895, thus making a total of 98 cases. Among this number the spleen was involved but three times. One of them, a cyst of the spleen, is in the possession of the Museum of the University of Pennsylvania, and is without a history. In another, reported by Ainsworth, there were cysts in the bladder and lungs, and in the lower extremity of the spleen a growth about the size of a small orange, which consisted of a firm fibrous capsule embedded in the substance of the organ. Its contents were caseous, much resembled bone-marrow in color and consistence, and in the midst of this was a single, large, empty collapsed cyst. The third case, one of Osler's Canadian ones, showed cysts in the stomach, omentum, mesentery, pelvis, and spleen. The spleen extended beyond the costal border, presented three small vesicles at the hilus, and contained a single cyst the size of a cocoanut, everywhere enclosed by spleen tissue.

Among the cases observed by Davaine, Böcker, Mosler, Madelung, Finsen, and Neisser (1731 cases), the spleen was involved but thirty-eight times. These statistics suffice to emphasize the infrequency of splenic involvement. The detailed accounts of the three American cases would suggest that this case is probably unique in so far as the United States

and Canada are concerned, in having involved the spleen alone of all the organs. It is possible that the University of Pennsylvania Museum case may have been limited to the spleen, and it is also possible that in the case in question hydatids may have existed in the liver at some earlier period and have escaped into the stomach or intestines, or, as in two cases reported by Fagge, in which recovery occurred, have passed through the diaphragm into the lung and been expectorated; but there were no postmortem evidences of any such termination. The only organ in which echinococcus-cysts occur that was not examined in this case was the brain. It is rarely involved, and it would in all probability have been explored had there existed any evidences of brain-lesion.

The echinococcus-cyst is the product of the tænia echinococcus which, finding its way into the alimentary tract of man, penetrates the stomach or intestinal wall, and is conveyed by the vascular system to some one of the viscera, where it becomes arrested in the capillary vessels. It may escape the capillaries by means of its hooklets. It soon ceases movement, sheds its hooklets, increases in size, and passes from a solid to a vesicular state, containing a transparent fluid. The surrounding tissues, as the result of irritation, become granular, and before long the parasite is surrounded by a distinct membranous sheath made up of connective tissue and well supplied with bloodvessels. This forms the permanent capsule. Sometimes at this stage, instead of going on to the development of daughter-cysts, the growth ceases and a sterile cyst remains. In these, or even in the normally developed cysts, inspissation of the contents—the so-called death of the cyst—may occur, and we have resulting a condition the counterpart of that illustrated in the specimen before you—a putty-like substance made up in large part of calcareous salts, which, in the case of the normal cyst, may contain hooklets and portions of the membranes of daughter-cysts, the whole enclosed in a fibrous capsule. *February* 11, 1897.

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5. *Amyloid degeneration of suprarenal gland.*

Exhibited by David Riesman, M.D.

The suprarenal gland from which the sections shown here were taken comes from the body of a man, about sixty years of age, who died of chronic tuberculosis, from which he had suffered for probably two and one-half years.

At the autopsy I found extensive caseation of the right lung, with

cavity-formation and firm pleural adhesions. The left lung was emphysematous, but free from tuberculous change. The liver and spleen were markedly amyloid; the suprarenal body was firm, not much enlarged, and had a peculiar grayish, translucent, or waxy appearance suggestive of amyloid degeneration. No abnormal pigmentation of the skin existed.

The patient was much emaciated and had suffered from dyspepsia, which, indeed, was his chief complaint, and he believed the cough was largely due to catarrh of the stomach. He was extremely feeble, and had not been out of the house or out of bed for several months before his death, always pleading lack of power and strength when urged to leave the bed. This extreme prostration, hardly accounted for by the unilateral tuberculous disease, may find a partial explanation in the degeneration of the adrenals. Vomiting was not present. The patient had suffered for many years from rumination, and was one of the cases reported by me in a paper on this subject.[1] Toward the last this symptom did not trouble him very much.

The suprarenal was fixed in 10 per cent. formalin-solution, hardened in alcohol, and stained with hematoxylin and with gentian-violet. On examining the section we find that the amyloid degeneration has attacked chiefly the cortical portion, but that the medullary is also to a considerable extent affected. Just within the capsule, in the zona glomerulosa, there are still visible well-preserved groups of polygonal cells, evidently epithelial, the nuclei of which stain darkly with hematoxylin. Fat, which is normally present in these, is nowhere discoverable. In places the cell-masses are destroyed, and the amyloid process extends directly up to the capsule.

In the zona fasciculata the amyloid disease is very extensive, and has given rise to homogeneous, not sharply defined, areas, taking a uniform pale-blue stain with hematoxylin. These areas seem to run in bands like the columns of the fasciculated layer. Between the amyloid portions many well-preserved parenchyma-cells are seen. They are recognizable by their round, darkly stained nuclei, which look somewhat like those of the liver-cells. Many connective-tissue cells and some nonstriped muscle-cells are also seen.

In the zona reticularis the amyloid change is less extensive, and the pigmented cells, some of which have large nuclei, are well preserved. A few pigmented cells are also seen in the zona fasciculata. The pigment is yellowish-brown, and is found in the protoplasm of the cells. Some of the pigmented cells are undergoing a degeneration, and have

[1] The Journal of Nervous and Mental Disease, June, 1895.

lost their nuclei. In the medulla the amyloid areas appear in spheric or oval masses, and are especially abundant near the large venous channels; many ganglion-cells are also seen.

Although, as you will see on examining the specimen, the amyloid degeneration is extensive, no bronzing of the skin existed; the mucous membranes were not examined. From what is known of the pathogenesis of Addison's disease, we would not expect it to occur in conditions of moderate amyloid degeneration, since that process affects the vascular structures, the connective tissue, and not the parenchymatous cells, which remain intact for a long time and perform the function of the gland, whatever it may be. In our section you will find a large number of fairly healthy parenchymatous cells, especially in the zona glomerulosa. Yet we may conceive the possibility of the amyloid change becoming so extensive that the parenchyma, pressed upon and deprived of its blood-supply, suffers total destruction, and that Addison's disease then develops. As the naked-eye changes are not very striking, they may be overlooked, the suprarenals considered normal, and the case recorded as one of Addison's disease without affection of the adrenals.

March 25, 1897.

VIII. MISCELLANEOUS.

1. *Microscopic examination of tapeworms.*

Read by Joseph McFarland, M.D.

I do not know that I am the first to employ the simple method about to be described, and have not gone into the literature beyond the ordinary laboratory hand-books to determine that point. It occurred to me one day, while attempting to secure a neat and transparent segment of one of the common worms, that there was no reason why the proglottides might not be treated as ordinary cut sections of tissue and examined in the same way. Rejecting, therefore, the recognized methods of maceration in caustic potash, and the unsatisfactory mounting in glycerin and similar reagents, I selected the flattest segments, dehydrated them thoroughly, and immersed them in xylol, benzol, and the various oils for clearing. After numerous experiments the following very simple method was found highly satisfactory:

The flattest proglottides are selected for examination. If they happen to be very fleshy, as is sometimes the case, it is best to compress them between glasses held together by rubber bands or springs before placing them in alcohol. Care must be taken that the pressure is not too great. After thorough dehydration in alcohols, 80, 95, and 100 per cent., and finally immersion for some hours in absolute alcohol, the segment is placed in fresh oil of cloves. In a few minutes the edges of the segment will begin to clear up, and in half an hour the tissues of the segment will be seen to disappear in the oil, leaving the branched uterus with its eggs conspicuous in the central portion. As the penetration of the uterus seems to be slow, a perfect specimen requires at least two hours' immersion in the oil. I have found twenty-four hours in the oil to be an improvement rather than otherwise.

After the dehydration and clearing the segment is placed upon a slide, touched for a moment with absorbent paper, then covered with a large quantity of balsam and a cover-glass. In absorbing the excess of oil with the absorbent paper care must be taken, for too much oil can easily be pressed from the proglottid, under which condition the removal of the blotter is followed by an absorption of air into the tissue, with a

subsequent vacuolated appearance of the proglottid that is unsightly and irremediable.

In properly prepared specimens the tissues are perfectly transparent throughout. The sexual opening is very distinct, the vagina and vas deferens can be seen entering the body cavity of the segment, the eggs can be seen in the uterus, and almost every organ of the segment is visible.

As the structure of the proglottid is not composed of nucleated cells, the use of nuclear stains was found to be disadvantageous rather than otherwise.

I exhibit to the Society preparations of proglottides of the tænia mediocanellata and bothriocephalus latus. *May* 13, 1895.

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2. *Filaria sanguinis hominis.*

Exhibited by Frederick P. Henry, M.D.

The patient from whom these living specimens of filaria sanguinis hominis nocturna were obtained was a woman who was born in Columbia, S. C., and had spent the first twenty-seven years of her life in that town. Her twenty-eighth year was passed in Palatka, Florida, and her twenty-ninth in Philadelphia, where she arrived in March, 1895. The symptom which first excited suspicions of filariasis was hematochyluria, which appeared on the third day after her delivery of a child at term. At various periods since that event this symptom has returned, and generally after some unusual exertion, such as lifting heavy weights, washing clothes, etc. The case is of unusual interest, because it is the first of the kind that has been observed in Philadelphia.

For further details of this case the reader is referred to the *Medical News* of May 2, 1896. *February* 27, 1896.

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3. *Tænia mediocanellata.*

Exhibited by M. H. Fussell, M.D.

The specimen was removed from a patient at the Medical Dispensary of the University of Pennsylvania.

The patient fasted for twenty-four hours, having previously taken ℥j

of castor-oil. At the end of twenty-four hours' fast, f℥iss of oleoresina filicis was given, followed in five hours by f℥j of castor-oil. The result was the specimen presented—the entire worm, including head. It measures ten feet, after having been soaked in alcohol for several days before it came to me.

It is a specimen of the beef-tapeworm, as shown by the minute head with four sucking disks, without any row of hooklets. The head is followed by the usual segment enlarged, followed by the tapering, slender neck, scarcely larger than a thread and terminating in the mature segments, which are longer than broad. The head and some of the immediately following small links are seen under the microscope, the rest of the worm between the glass. *March* 12, 1896.

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4. *Fetus of four and a half months.*

Exhibited by M. H. Fussell, M.D.

This specimen I present for Dr. Henry Jump, of West Philadelphia.

The patient was a primipara with good history. There were no unusual signs until two weeks ago, when the patient had severe abdominal pains, accompanied by rather severe bleeding.

The vague pains continued until last night, when severe labor set in without any increase of the bleeding. The os was long in dilating, but the fetus was suddenly expelled after the os was dilated.

The specimen is unique in my experience. It consists of the fetus enveloped in all its coverings, with the placenta in the exact position it occupied while in utero. The membranes are unruptured.

The placenta is lateral, and the lower end must have been very near the os. It is beginning to atrophy, and is not nearly as thick as the upper portion. Through the unbroken membranes can be seen the head, the cord, and the body with folded limbs.

The specimen is of further interest in showing the large proportion of the amnion attached to the uterus at this period. It occupies a full half or more of the entire surface. *March* 12, 1896.

5. *A case of pulmonary tuberculosis.*

Exhibited by M. H. Fussell, M.D.

I owe the privilege of presenting these specimens to Dr. John Woodruff, of Roxborough. I never saw the patient alive, but made the sections.

The case was diagnosed as one of typhoid fever, and a review of the symptoms will show how readily such a diagnosis could have been made; indeed, it seems the only diagnosis that was justified. In view of this fact and the added one of the prominence of brain-symptoms, it is greatly to be regretted that an examination of the brain could not be made, but this was denied us.

Robert D., aged fifty-three years, marble-cutter; alcohol moderately. Family-history unknown. His previous health, with the exception of the usual diseases of childhood, has been good.

March 10, 1896. Stated that he had felt languid, had occasional headaches and dizziness, nausea, constipation for the past two weeks. About three days ago he had a chill lasting about one hour, which was followed by fever, and trouble with vision (saw stars), also since then he has had considerable pain when at stool. Two weeks ago drank beer with a typhoid patient, using the same glass.

Examination. Tongue coated and brown and indented; abdomen very tense; spleen not palpable; pressure in right inguinal region elicits gurgling tremulous condition of the whole body. When talking the sentences are somewhat disconnected.

11*th.* Tongue still coated and much swollen; passed a sleepless night; intense frontal headache, and five to ten minute paroxysms of coughing at half-hour intervals; temperature 103.4°; pulse 97; respirations 42. Muttering delirium developed; spots on chest and abdomen, resembling the typhoid eruption, appeared, also some small red papules. Voided urine and feces involuntarily. Cheyne-Stokes respiration; muscular twitchings throughout the whole body. Soon coma ensued, from which he passed into death. Morning temperature 101.6°; pulse 150; respirations 63. Evening temperature 100°; pulse 150; respirations 60.

Postmortem. Considerable adipose tissue, not much emaciation; body well covered with hair; several red papules over body, and two very small petechial spots in the left parasternal line; abdominal wall very thick; much adipose tissue. A cartilaginous union between the fifth

and sixth and between the seventh and eighth ribs at junction of ribs with the cartilage on right side.

Thoracic cavity. Lungs had a marbled appearance. There were some old adhesions at the apices of both lungs; none at bases. No liquid in either pleural cavity. The level of the diaphragm on the right side was opposite the fourth rib, and on the left side opposite the fifth rib. Considerable fat in the anterior mediastinum. The left lung was crepitant throughout, except at the extreme apex, which was the seat of a semicalcareous deposit; the base showed hypostatic congestion; the pleural surface at the base contained sparsely scattered miliary tubercles, which were also seen on the cut surface. The right lung showed the same condition as the left one, except that the consolidation was of greater extent, and there was one cheesy mass at the apex. The costal surface of the pleura was smooth to the touch on both sides.

Heart. The pericardium contained the normal amount of fluid. The left side of the heart was collapsed; the right side was distended, and occupied almost the entire anterior aspect of the heart. On the upper surface of the right ventricle was a white fibrinous patch, U-shaped, measuring two by one-and-a-half inches, which proved to be in the pericardium. On opening the right cavity the right ventricle was filled with fluid blood and jellified and chicken-fat clots. A large clot filled the superior vena cava; the right auriculo-ventricular orifice easily admitted four fingers and thumb; the tricuspid valve was thickened, especially the posterior leaflet; the pulmonary semilunar valves were normal; the walls of the ventricle were thin and the cavity dilated. The left side was well contracted; the mitral orifice admitted three fingers, and the valves were about normal. A small chicken-fat clot found in the aorta. Aortic valves were normal. There were numerous small atheromatous plates found in the aorta, which was also slightly dilated. The walls of the left ventricle were soft.

Abdominal cavity. The intestines were not distended, and there was about the normal amount of fluid in the cavity; the sigmoid flexure occupied the greater portion of the hypogastrium; the appendix was long, normal, and pointed posteriorly; there were no miliary tubercles over the peritoneum; there was a Meckel's diverticulum in the lower portion of the ileum; in the jejenum were a number of small, dark masses, varying in size from a pin-head to a split pea; they were hard and rounded, and on section proved to be thrombi in the course of the vessel. As the ileum was approached they became much less numerous, and as the valve was reached they had disappeared entirely. Neither Peyer's patches nor the solitary glands were enlarged, and could only be

observed with difficulty. There were no ulcers in the cecum; the urinary bladder was partially filled with fluid.

Spleen was very small and soft, and contained no tubercles.

Stomach was dilated and contained a small amount of well-digested food. The esophageal and pyloric orifices were free, and there was no ulceration. Gall-ducts were perfectly clear and free. Pancreas was thickened and much hypertrophied, but not the seat of any new growth.

Liver was about normal size, and over the right surface were a few opaque spots (not nodules), but on section were seen embedded in the liver-substance. Right kidney normal in size, and contained several fatty nodules on the surface of capsule. The capsule was adherent and the cortical substance comparatively small. Left kidney normal in size, rather hard, and capsule was adherent; the cortical and medullary substances were in about proper proportions; the suprarenal capsules were softened, and seemed to contain a cavity, but were not the seat of any new growth. *March* 26, 1896.

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6. *Foreign body (remnants of bone) forced through the craw of a turkey.*

Exhibited by F. Savary Pearce, M.D.

This specimen was handed to me by a lady asking for information as to the protrusion here seen on the anterior outer surface of the craw of a turkey. Her experienced cook had seen nothing like it before, and had hacked the specimen much in investigation of it. On close inspection, however, there is seen an opening into the detached segment of the thick muscular wall, quite removed from the esophageal and intestinal openings. Through this opening the probe can be carried from the mucous membrane in a curved direction up and out a distance of two and one-half inches, where it strikes upon a sharp body of irregular shape and measuring five-eighths by one-quarter inch. This seems to be inspissated food-stuffs with a nucleus of jagged bone-salts. The gristly canal is filled with food-stuffs.

The explanation that seems plausible is of the swallowed foreign body being forced ino the muscular wall by the powerful contractions. This gradual forcing on has produced no signs of active inflammation. It might in time have been expelled into the peritoneal cavity.

Dr. Harrison Allen saw the specimen, and informs me that he is aware of no such anomalous diverticula in the fowl, and thinks the explanation given is the only rational one. The specimen is interesting as showing

the jaw-like force exerted by the craw-muscle, and as demonstrating the immunity of tissues to foreign bodies under favorable circumstances.

In 1892 I presented a specimen to this Society of a portion of liver of a chicken transfixed by a pin, which the late Dr. Formad placed in his collection at the University. In the discussion Dr. Stengel remarked (recalling Dr. Allen Smith's specimen) that in human beings so important organs as the heart and pericardium may be so pierced without necessarily producing symptoms. *April* 9, 1896.

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7. *The theories which have been advanced to explain immunity from infectious diseases.*[1]

Read by B. Meade Bolton, M.D.

It is hardly necessary for me to say that at present there is no one exclusive theory of immunity that has found universal adoption. If there shall ever be found a theory that will fit all cases it remains for future discovery. It is at present impossible to dogmatize. Conflicting ideas have been so ably advanced and ingeniously attacked that judgment must remain suspended. But, although the theories themselves have not been proved, it stands very differently with the observations upon which the theories have been formed. There is a long array of very definitely determined facts in regard to the laws of immunity. Whatever the reasons may finally turn out to be, there are certain phenomena which always follow upon certain conditions with absolute regularity. It will be my endeavor this evening, after stating each theory, to give very briefly some of the more convincing facts that have been determined beyond dispute, both in support of and opposed to the theories. The literature of the subject is very large and interesting, and I have tried to cull out some of the more important parts as an aid to those who are not able to spare the time to read up the subject for themselves. I shall give the theories more or less in chronologic order.

The theories which have been advanced to explain immunity from infectious diseases, and which have more or less experimental evidence to support them, are the following: 1. The exhaustion-theory (théorie de l'équisement, of Pasteur). 2. The accumulation-theory (théorie de la substance ajoutée, Chauveau). 3. The accommodation-theory, Behring.

[1] Read at the Semi-annual Conversational Meeting, April 23, 1896.

4. The theory of special organs, Brieger, Kitasato, Wassermann, *et al.* 5. The humoral-theory, Behring. 6. The cellular-theory: (*a*) the starvation-theory of Grawitz; (*b*) the phagocytic-theory of Metschnikoff. 7. Theory of alexins, Hankin. 8. Theory of special enzymes of Pfeiffer.

1. The exhaustion-theory is the first one advanced to explain the reason why recovery from an attack protects the indvidual from subsequent attacks of the same disease, and it has recently been advanced as an explanation of natural immunity. According to this theory there is either a natural or acquired absence of substances necessary for the growth of the pathogenic micro-organism of the disease in question. In smallpox, for example, all of the cells of the epidermis which are suitable for the growth of the smallpox-organism, whatever this may be supposed to be, are destroyed by an attack of the disease, and only those cells remain and propagate that are unsuited for the growth of the smallpox-organism. These cells transmit to their offspring this characteristic. In the case of other diseases it may be some chemical substance contained in the animal body which becomes exhausted by an attack of the disease. In support of this view Pasteur cites the fact that if a micro-organism is grown in bouillon it sooner or later exhausts the nutrient material, and the bouillon no longer serves as a medium for the growth of the micro-organism in question. Pasteur took the micro-organism of chicken-cholera, and having grown it for four days in the incubator found that all growth had ceased. He then sterilized this bouillon and inoculated it with the chicken-cholera organism, but obtained no growth. He concluded from this that the original growth of the organism had exhausted the nutrient material in the bouillon, and that in like manner the growth of micro-organisms in the animal body also exhausts the nutrition for the organism in question.

The latter deduction is, of course, not justifiable, the conditions in the animal body being totally different from those existing in the experiment. Nevertheless, this same view has been recently advocated as an explanation of natural immunity. That is to say, that certain races of animals and certain individuals have immunity because some substance necessary for the growth of the organism is normally absent from their tissues.

There are many difficulties in the way to the acceptance of this theory. If the tissues of one of the immune animals are cut up and used as culture-medium the organisms will grow perfectly well upon the medium, showing that the nutrient materials are present. Then, again, the animals that enjoy immunity have the power of bearing inoculations

with the poisonous products of the micro-organism. In this case the growth of the micro-organism in the tissues has nothing to do with the production of the disease. On the contrary, it may be that the micro-organism will grow in the tissues and yet not produce the symptoms of disease.

This theory is, therefore, quite generally abandoned, but will always deserve mention on account of the advocacy of Pasteur.

2. The accumulation-theory of Chauveau, although in its original significance it has shared the same fate as the exhaustion-theory, is perhaps, in a way, nearer to the truth. The original argument in support of this theory is really only a different interpretation of the Pasteur experiment. It is claimed that the micro-organism fails to grow in the medium inoculated a second time, not because the nutrient material is exhausted, but because there is an accumulation of the products of growth of the micro-organisms. There is an accumulation of the bacterial excreta, so to speak. Thus, in alcoholic fermentation the yeast-cells cease to grow after a certain percentage of alcohol has been produced. Further growth is prevented by the alcohol, even if there is still an abundance of nutrient material present. And so Chauveau supposed it occurred in all animals attacked by disease, viz., an accumulation in the animal body of products that make the tissues unsuited to the growth of the micro-organisms.

It is hardly necessary to dwell at length upon this theory; it has been quite abandoned. The saturation of the tissues of the convalescent animal with effete products of growth does not explain the phenomena of immunity, and it has been pointed out that material of this kind could not be stored for any length of time in the body, but would be quickly eliminated.

An accumulation-theory that has greater plausibility in its favor is that in every culture there exists, side by side, the poison and its antidote, and that in inoculating animals with cultures of the micro-organisms the antidote accumulates and the poison is eliminated. This theory even has some experimental facts in its support, and it seems difficult to overthrow it entirely. It will come up again in the theory of special organs, where it is claimed that the thymus gland has the power of depriving cultures of toxic properties and allowing the antitoxic properties to operate. If cultures are treated with various chemical substances or with heat, or with weak electrical currents, they are found to contain substances that render animals immune on inoculation. It has been argued, however, in explanation of these facts that it is the toxin itself which has been changed in some way into its own antidote by the action of the various agents employed.

In the exhaustion-theory and the accumulation-theory the part played by the animal body is purely negative. The more recent theories that follow all take into account a participation on the part of the animal. The views in regard to the action of the tissues, and just what tissues are involved, differ widely, but all are agreed that there is a reaction on the part of the animal; the animal cannot be regarded as a merely passive object.

3. The theory of accommodation seems really to explain very little. It merely states that the normal or acquired tolerance to bacterial poisons is analogous to the normal insusceptibility (to certain poisons, exhibited by some animals) and the acquired tolerance shown by man to various drugs, as in arsenic or morphin-habitués. It is pointed out that goats may be given enormous doses of morphin, and many examples are quoted where animals are capable of withstanding inoculations of large amounts of bacterial poisons, as the tolerance of chickens for tetanus-toxin. The well-known tolerance of mice for diphtheria-toxin can also well be quoted as an example of natural tolerance. This tolerance is very remarkable. I have injected as much as 1 c.c. of diphtheria-toxin into a white mouse without producing any disturbance in the health of the animal, although less than one-tenth of a c.c. of the same toxin was certainly fatal to adult guinea-pigs in forty-eight hours. In fact, I have not found it possible to kill a mouse with diphtheria-toxin; for if they do not die directly after the injection from the shock of the comparatively enormous amount of fluid injected, they seem to suffer no inconvenience. The animal will run about, apparently in perfect health, with a tumor-sac filled with an amount of toxin that would cause severe symptoms, if not death, in a horse.

But, after all, it does not explain the matter to assign as a reason the unknown cause of an analogous process. Moreover, the two processes are not strictly analogous, for it has been pointed out that animals gradually injected with increasing doses of a bacterial poison become tolerant of the poison much more quickly than can be accounted for on the ground of gradual accommodation. It has been pointed out that there is a more or less distinct crisis in the inoculation of animals with bacterial poisons similar to the crisis of pneumonia. The susceptible animal withstands small doses at first with more or less reaction, and then more or less suddenly resists without reaction comparatively large doses. Indeed, it is claimed that in the production of antitoxin it is necessary to produce more or less of a crisis. But it cannot be denied that the gradual accommodation of the cells to poison may operate along with other processes, although it alone is insufficient to explain all the phenomena of immunity. It offers no explanation of the production of

antitoxin, and yet this must be intimately associated with the production of immunity.

But the accommodation-theory begins to approach somewhat nearer the truth, for it recognizes at any rate that the animal tissues take some part in the process.

4. The theory of special organs is based upon analogy and upon experimental evidence. It claims that the function of immunity is due to some special organ or organs analogously to the other functions of the body. In other words, the tendency to ascribe special work to special organs has led to the conception that there is a special organ or set of organs for the purpose of warding off the attacks of bacteria. For several reasons it was supposed that the thymus gland possessed this function, but it has also been attributed to the spleen, the liver, the thyroid gland, and the lymph-glands. The manner in which these organs may operate is variously explained. It is held by some that the property is due to the juices secreted by the cells; by others that it is due to the cells themselves. The endothelial lining of the bloodvessels has also been supposed to destroy the bacteria circulating in the blood.

In spite of the fact that the thymus gland is a temporary organ and that the phenomenon of immunity exhibits itself more markedly in adult animals than in very young animals, nevertheless the experiments made to test the action of the thymus gland gave very positive evidence of its effects. The results also seem to substantiate the view that the immunizing substance is contained in cultures of the pathogenic bacteria along with the disease-producing substance. The effects of the thymus gland can be demonstrated in two ways: either by growing the bacteria to be tested on aqueous extracts of the gland or by mixing an aqueous extract of the gland with cultures of the bacteria. If the thymus gland is chopped up and an aqueous extract made of it, the bacteria grow perfectly well, but such cultures possess little or no pathogenic properties. If this aqueous extract is mixed with cultures grown in some other medium, it deprives the latter of much of their pathogenic property. But, more than this, cultures of bacteria grown in thymus extract or mixed with thymus extract can be used successfully in producing immunity in the animals inoculated. It has been claimed that the action of the thymus extract is to deprive the culture of the disease-producing element and leave the protective element intact. It is supposed, in like manner, that the thymus gland while in the body has the power of neutralizing the pathogenic properties of the bacteria circulating in the blood, and that then the protective element produced by the bacteria comes into play.

In support of the view that the lymph-glands are a source of protection the fact is pointed out that in cases of infectious wounds the neighboring lymph-glands become enlarged, and that the infection often stops short there. And, in like manner, there is more or less evidence for the action of the spleen and liver, as well as for the endothelial cells.

But this view of immunity is insufficient to explain all the phenomena, and cannot be regarded as the sole cause at any rate.

5. The humoral-theory and the following cellular-theory, are the two that practically divide the scientific opinion of the day. Broadly stated, the humoral theory attributes to the liquid portions of the blood the capacity to neutralize the pathogenic properties of the bacteria and to kill the bacteria themselves. The cellular theory, or the phagocyte-theory, attributes this to the white blood-corpuscles, or to substances derived from them, and there is a great deal of evidence in favor of both of these theories. It is true, on the one hand, that the liquid portions of the blood destroy the bacteria and in immunized animals neutralize the poisonous properties of the bacteria; and it is also true, on the other hand, that the white blood-corpuscles destroy the bacteria.

These two facts are abundantly proved and can be no longer disputed for particular cases.

If, however, immunity were due to the germicidal properties of the blood-serum alone, there would have to be a correspondence between this germicidal action on the one hand and the immunity of the animal on the other. But no such correspondence exists. It has been shown that the serum of the blood of a highly susceptible animal has the power of destroying the bacteria of the disease in question. It has also been shown that the blood-serum of an animal that is refractory to a disease may show no germicidal properties. A small amount of the blood-serum from a rabbit will kill enough anthrax-bacilli in a culture to produce disease and death in many rabbits. The blood-serum of dogs has little germicidal power upon anthrax-bacilli, yet this animal enjoys a high degree of immunity. So there are insuperable difficulties to the acceptance of the humoral-theory as the sole explanation of immunity.

6. The phagocyte-theory has received more attention both from its advocates and from its opponents than any other theory, and the amount of literature upon the subject which has rapidly appeared is very large. Nevertheless it is possible to state briefly the reasons advanced for and against the theory. It has been most ingeniously defended and attacked; the same identical observations have been used as arguments in favor of the phagocyte-theory and against this theory.

It has had to be conceded even by the most active opponents of the phagocyte-theory that the white corpuscles take up the bacteria not only in cold-blooded animals, as was at first claimed, but also in other cases. But the opponents recognize in this only a process of elimination of dead foreign particles. They claim that the micro-organisms that are taken up in this way are first killed by the fluids of the body, and that the leukocytes take them up just as they were long known to take up any floating particles; that there is no special action on the part of the leukocytes to destroy the living bacteria. Flügge says the leukocytes produce the impression of tombs appearing behind a line of battle after the conflict is over, and the phenomenon of Pfeiffer would seem to establish this view most conclusively in the case of guinea-pigs in which immunity from cholera has been artificially produced. Pfeiffer's phenomenon has been abundantly corroborated, and Metschnikoff himself pays Pfeiffer a high compliment for having observed it. The phenomenon as observed by Pfeiffer and others is that the cholera-bacilli introduced into the peritoneal cavity of immunized guinea-pigs quickly become distorted, assuming for the most part globular shapes (arthrospores of Hueppe). These forms are usually interpreted as forms of involution immediately preceding disintegration. If a drop of the peritoneal fluid of an immunized guinea-pig is examined within half an hour or more after virulent-cholera bacilli have been injected, it will be found to be devoid of leukocytes, and it would appear impossible for these to have taken any part in the process up to this point. Even the leukocytes found normally in the peritoneal fluid have entirely disappeared. The fluid that was at first slightly turbid from the presence of leukocytes has become clear and transparent. This is the stage of hypoleukocytosis, and is followed by a great increase in the number of leukocytes that soon become filled with the distorted cholera-bacilli, the stage of hyperleukocytosis. So stated, the conclusion would seem to be inevitable that the fluids of the peritoneal cavity cause the death of the cholera-bacilli and that the leukocytes then remove the dead bodies of bacilli, and yet it can be shown that quite a different interpretation is permissible and even plausible. Metschnikoff succeeded without difficulty in obtaining Pfeiffer's phenomenon, but he does not see that the application of the phagocyte-theory in explanation would be totally "erroneous," as Pfeiffer states. On the contrary, while he admits that it is theoretically probable that there are exceptions to the general rule, the examination in detail of the phenomena of extracellular destruction of bacteria shows that this does not belong to the category of exceptions. By a study of the peritoneal fluid of an im-

munized guinea-pig inoculated into the peritoneal cavity with a culture of cholera-bacilli, Metschnikoff observed the following sequence of events. Preparations made in from one to five minutes after inoculation show the large mononuclear and polynuclear leukocytes all in a state of repose, or at most a few with weak movement and well preserved. The most of them appear very abnormal, massed together in larger or smaller clumps lying in a glairy fluid. All these changes terminate in the dissolution of certain leukocytes and the disappearance of the masses which have been formed and have attached themselves to the surfaces of the abdominal organs. There takes place a veritable "phagolysis" affecting only the true phagocytes—*i. e.*, polynuclear and mononuclear leukocytes and eosinophiles; the lymphocytes are not affected. Although the stage of phagolysis is of short duration—only about half an hour—it serves to explain the striking appearance of a number of leukocytes literally surrounded by the micro-organisms, but unable to take them up, and finally the disappearance of the leukocytes. After this period the normal leukocytes again appear; first the lymphocytes, afterward in association with polynuclear leukocytes. Then follows at once the period of phagocytosis, and the peritoneal fluid becomes cloudy and soon is found to contain neither normal bacilli nor free granules. Nothing can be observed but granules within the leukocytes. If some of the fluid is transferred to peptonized water or agar-agar, some of the bacilli will grow even after ten or fifteen hours or more, showing that the leukocytes have taken up living bacilli.

Three stages, then, can be noticed: 1. Abnormal appearance, and finally dissolution of the leukocytes present in the peritoneal fluid—*i. e.*, phagolysis; 2. The phenomenon of Pfeiffer, in which the bacilli become distorted by the action of the peritoneal fluid. This action is due to a substance given off by the fixed cells of the peritoneum, according to one view, and due to substances given off by the leukocytes, according to another view; 3. Phagocytosis.

It is possible completely to eliminate the first two stages and to bring about the process of phagocytosis pure and simple; to prevent the occurrence of Pfeiffer's phenomenon, yet to observe that the organisms are destroyed just as promptly by phagocytosis alone. If twenty-four hours previous to the inoculation with cholera-bacilli the animal is injected into the peritoneal cavity with 3 c.c. of ordinary sterilized bouillon, the leukocytes are preserved, and the inoculation with cholera-cultures does not produce hypoleukocytosis. The stage of hyperleukocytosis begins at once. The organisms do not become distorted outside

the leukocytes, but are taken up without alteration, and many are found with their normal shape inside the leukocytes, where they finally become distorted.

It would really seem that Metschnikoff had never found better evidence for his doctrine than in the results obtained by inoculating cholera-cultures into the peritoneal cavity of immunized guinea-pigs. Pfeiffer has recently acknowledged that in similar inoculation of guinea-pigs with typhoid cultures there is extensive phagocytosis.

A different view has been advanced by Grawitz to explain the manner in which the leukocytes destroy the bacteria. According to this view, the leukocytes do not actively destroy the bacteria, but simply deprive them of nutrition. But this can hardly stand in the light of evidence in favor of actual phagocytosis.

7. Hankin's theory of protective proteids is based upon the observation that extracts of a proteid obtained from the pulp of the spleen of rats are capable of killing anthrax-bacilli. But the results of others show that this is not a universal rule, and, although it may be in some cases one of the means of defence of certain animals, it cannot be regarded as an explanation of immunity.

Hankin's original statement was to the effect that the disintegration of the eosinophiles liberated the alexins. In a subsequent paper he states that he did not mean the eosinophiles, but the neutrophiles. This brings Hankin's theory much in accord with Metschinkoff's view of phagolysis.

8. Pfeiffer's theory of special enzymes is based upon observations made on cholera and typhoid fever. According to Pfeiffer, neither the theory of the production of antitoxins nor the phagocyte-theory is capable of explaining the immunity in these diseases. The inoculation of animals with gradually-increasing amounts of cholera-culture, even where a very large degree of tolerance has been attained, does not produce a substance that will neutralize the poison in old, dead cultures. The serum of animals gradually inoculated possesses high germicidal value, but little antitoxic properties. The normal blood-serum of goats has a certain degree of antitoxic value, and the serum from inoculated animals has but little more. But the serum of inoculated animals has a much higher germicidal value than the serum of uninoculated animals. This fact makes it impossible to explain the immunity acquired by inoculation of cholera-cultures by the theory of antitoxin. The production of antitoxin in tetanus and in diphtheria is admitted.

Pfeiffer also finds objections to the phagocyte-theory. He fails to

observe in his phenomenon already alluded to any evidence of leukolysis or phagocytosis. Moreover, if pus from an abscess in the animal is used, instead of serum, his phenomenon is not as pronounced. But if the phenomenon is produced by the substances derived from the breaking up of the leukocytes, surely it would take place best in pus or in an abscess. In his last publication, which appeared after the above was written, Pfeiffer acknowledges phagocytosis in the peritoneal cavity of guinea-pigs inoculated with typhoid cultures, although he states that there is also extracellular destruction as well.

These are the main points of his objections to accepting either the antitoxin-theory or the phagocyte-theory, and consequently he seeks another explanation and finds it in the theory of special enzymes. This theory is a very attractive one, and explains many of the phenomena that cannot be fully explained by any of the other theories. According to this theory, the blood of an animal that has been gradually inoculated with increasing quantities of dead cholera-cultures contains two substances: It contains a certain substance that can be converted into a germicide, and in addition it contains a certain amount of the ready-formed germicide. The former is capable of being converted into the latter by certain enzymes in the animal body, and is called the immunity-substance in contradistinction to the fully-formed germicidal substance. An illustration will serve to make this clear. If the blood-serum of an animal that has been gradually inoculated for some time be tested, it will be found capable of killing a greater or less amount of cholera-bacilli in a culture, and also of protecting animals from the effects of virulent cholera-cultures. If this same serum is heated at 60° C., it is deprived of the power of killing the cholera-bacilli in cultures, but is not noticeably affected in its power of protecting animals. Its germicidal value is destroyed, but its immunizing properties are retained to their full extent. The substance that remains after heating the serum has neither germicidal nor antitoxic properties itself, but develops germicidal properties in the animal injected with it. It is supposed to be stored up in the body and changed into the germicide by a fermentative process of the cells of the animal whenever the animal is inoculated with the virulent cultures of cholera. It is supposed that the animal cells contain enzymes which bring this about. Animals yield this immunity-substance actively by injections of dead cholera-cultures, or they may be the passive recipients of the substance formed in another animal. Whether the animal has formed it actively himself or whether he has received a dose of it that has been formed by some other animal, in either case it is converted into the germicide when the

animal is injected with virulent cultures. The germicide is only formed as occasion requires.

This theory explains perfectly the more or less sudden production of immunity as observed in the crisis in some diseases.

The theories above and the prevalent attitude that has been assumed in regard to them may be summarized as follows:

The theory that immunity is due to the natural absence of necessary food or to the exhaustion of all the necessary food by an attack of the disease is generally abandoned.

The theory of the accumulation of products noxious to the bacteria is also abandoned, at least in its original conception.

The theory that the tissues gradually become habituated to the presence of the bacteria and bacterial poisons is abandoned, for the reason that immunization often takes place too quickly for this, and besides it does not explain the occurrence of a crisis.

The theory of special organs as the seat of the destruction of bacteria and the neutralization of toxins has not sufficient evidence in its favor, and, moreover, the bacteria and their toxins are destroyed outside of the organs in the fluids of the body.

The theory of alexins produced by the neutrophilic leukocytes has not had sufficient evidence in its favor to command any great following.

The theory of special enzymes is so recent that it is impossible to say whether it will stand the test of criticism and further observation.

The theory that the leukocytes deprive the bacteria of food, and thus starve them, cannot hold good, because the bacteria will grow perfectly well in artificial media made from the tissues of the immunized animal. There remain the two theories—the humoral theory, on the one hand, and the cellular theory, on the other—the one claiming for the fluids of the body the capacity for destroying the bacteria and neutralizing the bacterial poisons; the other claiming that the bacteria are devoured by the polynuclear and large mononuclear leukocytes. Both of these are ably supported and both have been seriously attacked.

The question as to whether we shall finally adopt one or other of all the theories alluded to above as an explanation of immunity must at present remain in doubt. It must also remain questionable whether it is possible to explain immunity by any one theory, or whether there may not always be many factors involved which cannot be made to fit under any one exclusive theory. However this may be, it is at least demonstrated that the body is naturally supplied with certain recognized weapons of defence and that it may acquire still others. We know that there are certain portals of entry by which the bacteria get

into the body and that these portals have natural guards. If the natural guards are weakened or destroyed and the bacteria gain entrance, they then have to contend with the germicidal action of the fluids of the body and with the leukocytes, and probably also with the fixed cells of the body. If the bacteria are able to overcome the action of the fluids and cells of the body, they produce death. If the animal tissues gain the upper hand, the individual not only recovers, but is protected against subsequent invasion for a longer or shorter time. Not only this, but there is an over-production of the substance, whatever it is, that has caused recovery, whether it be a germicide in the strict sense and destroys only the bacteria themselves, or whether it be a substance that neutralizes the products of growth of the bacteria.

It is no longer a mystery why we do not all contract infectious diseases. A great deal is known about our means of defence. The presence of the organism is an essential factor, but there are other equally necessary conditions for the production of disease. The natural guards at the portals of entry must first be passed, and the resistance of the fluids and cells inside the body must then be overcome.

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September 23, 1896.

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8. *Charcot-Leyden crystals.*

Exhibited by David Riesman, M.D.

In May last a patient came under my care who was suffering from a severe paroxysmal cough, setting in, as a rule, during the night. He was a robust, healthy-looking tinsmith, and presented nothing abnormal on examination, save that the chest was slightly emphysematous in shape. He had been ill but a short time, and previously his health had always been good. The sputum was scanty, tenacious, grayish-white, somewhat frothy, and had a faint sweetish odor. In two out of three specimens examined in the fresh state I found peculiar, transparent, somewhat shining crystals, composed of two sharply-pointed pyramids joined base to base. The crystals varied considerably in size, and were distributed irregularly through the sputum—some fields would show them aggregated in immense numbers, while in others but few isolated specimens could be found. Their edges were sharp, and

the ends acutely pointed; with few exceptions, they were nearly all perfect in outline. The crystals found were those commonly termed Charcot-Leyden, Charcot-Robin, or Charcot-Neumann crystals, the presence of which in the sputum has by some writers been looked upon as characteristic of asthma, a view that is, however, no longer tenable. The same sputum also contained, on one examination only, a few small crystals that I took to be calcium oxalate. On looking over the literature I find that the presence of calcium oxalate in sputum has already been noted by Ungar.[1]

Eosinophile cells were present in the sputum in abundance, and also pigment-cells enclosing coal-dust. The sputum contained no Curschmann's spirals and no tubercle bacilli. On standing, at room-temperature, the number of crystals increased noticeably.

In a more typical case of asthma, a boy, aged fifteen years, who was said to have suffered since the first year of his life, I also found large numbers of Charcot-Leyden crystals and several beautiful spirals, but in these a distinct central thread could not be seen. This sputum behaved peculiarly on standing: after a few hours large, transparent, flat plates, resembling cholesterin, appeared, but they were not superimposed upon each other in the manner characteristic of the latter crystals. After a few days the sputum no longer contained Charcot-Leyden crystals, but instead sheaf-like crystalline aggregates that resembled calcium phosphate.

Charcot-Leyden crystals appear to have been first seen in sputum by Charcot[2] in 1856, in a case of *catarrhe sec* with emphysema. Similar crystals had been observed by him and Robin,[3] in 1853, in the spleen and the blood of the right ventricle of a case of leukemia, and later by Charcot and Vulpian[4] in the same disease. Neumann[5] also found them in the blood of a patient dead of leukemia, and, at a later period, in leukemic and normal bone-marrow. Lewy[6] found crystals resembling those of Charcot-Leyden in polyps and papillomas of the nose; Förster[7] found them in a myxomatous tumor of the optic nerve and in the thickened mucus of a dilated gall-duct; and Leyden[8] in the sputum of asthma and, more recently, in the nasal mucus of a patient with acute

[1] Ungar. Quoted by Hoffmann, Specielle Pathologie und Therapie. Nothnagel, xiii. 3, 1.
[2] Charcot. Gaz. hebdom., 1860, p. 755.
[3] Charcot and Robin. Gaz. méd. de Paris, 1853, p. 430.
[4] Charcot and Vulpian. Gaz. hebdom., 1860, p. 755.
[5] Neumann. Quoted by Leyden in Virchow's Archiv, 1872, 54, p. 324.
[6] Lewy. Berlin. klin. Wochensch., 1891, 33, p. 816.
[7] Förster. Quoted by Leyden, loc. cit.
[8] Leyden. Virchow's Archiv, 1872, 54, p. 324; Deutsche med. Wochensch., 1891, p. 1085.

rhinitis, in whom neither nasal polypus nor asthma existed. Cohn[1] observed them, among other places, in the lymph-glands in malignant lymphoma, in mucous polyps of the nose, and in the bone-marrow of cases that had died of chronic nephritis and of pulmonary tuberculosis. Leichtenstern[2] records their presence in the feces of patients suffering from anchylostomiasis.

It is thus seen that the crystals are found in a great variety of conditions and places, which, perhaps, it is not superfluous to name, seriatim, as completely as possible. They have been found in the blood in leukemia, usually only after standing, but at times (Pruss[3]) in the fresh blood and the fresh juice obtained from the spleen (Westphal[4]); in the organs in leukemia, particularly the bone-marrow and spleen; in the sputum in asthma, pneumonia, emphysema, and various forms of bronchitis; in the feces of some forms of diarrhea, in cases of anchylostomiasis, and in typhoid fever (Nothnagel[5]); in the lymph-glands in malignant lymphoma; in bone-marrow in the normal state, and in cases of chronic nephritis and pulmonary tuberculosis; in the nasal mucus in rhinitis; in myxomatous tumors of the nose and optic nerve, in papillomas of the nose, and in thickened mucus of the bile-ducts.

The appearance of the crystals I have already described. In form they have always, until recently at least, been considered octahedra, but Cohn[6] has conclusively demonstrated that they are in reality hexahedra. He did this by hardening the sediment of the sputum obtained by means of a centrifuge, and cutting it into sections. In the latter he found many crystals cut transversely, and could readily count the number of sides. He also instituted a series of measurements; the longest crystal was 0.075 mm. long; the greatest width found was 0.04 mm. The value of the apical angle was from 17° to 20°.

Crystalloptic studies, made by him in conjunction with Hecht, gave additional proof that the crystals were not octahedra.

The chemic nature of the crystals is not definitely known. Salkowsky[7] long ago thought they were a crystallized mucin-like substance. Later writers identified them with Böttcher's sperma-crystals, which, chemically, consist of phosphoric acid and an organic base, known as Schreiner's base or spermin, C_2H_5N, or $C_{10}H_{26}N_4$ (?). Schreiner's phosphate, ac-

[1] Cohn. Arch. für klin. Med., 1895, 54, p. 515.
[2] Leichtenstern. Quoted by von Jaksch, Klinische Diagnostik, 4th German edition, 1896, p. 273.
[3] Pruss. Quoted by Westphal, in Deutsch. Arch. für klin. Med., p. 614.
[4] Westphal. Deutsch. Arch. für klin. Med., 1891, p. 614.
[5] Nothnagel. Quoted by von Jaksch, loc. cit.
[6] Cohn. Loc. cit.
[7] Salkowsky. Virchow's Archiv, 1872, 54, p. 344.

cording to Ladenburg,[1] has the composition $(C_2H_5NH)_4Ca(PO_4)_2$. But the crystallographic studies of Cohn have proved that Charcot-Leyden and Böttcher's crystals are not identical; aside from this, they differ also in their simpler physical characters, Böttcher's crystals being usually spindle-shaped, with curved planes and blunt edges. Crystalline conglomerates and twin forms are also more common than in the case of Charcot-Leyden crystals. The chemic nature of the latter remains, therefore, still to be solved.

Charcot-Leyden crystals are insoluble in cold water, ether, alcohol, xylol, chloroform, creosote, and solutions of iodin; they are soluble in warm water, hydrochloric, nitric, sulphuric, phosphoric, acetic, lactic, oxalic, picric, and carbolic acid, potassium, sodium, and ammonium hydrate.[2]

In asthma, leukemia, and other conditions, they are associated with eosinophile cells, and, according to Leyden, there is an intimate relation between the two; indeed, he believes that the crystals arise from the cells. The crystals in asthmatic sputum are larger if the attack has lasted a few days; it should, however, not be forgotten, as Hoffmann points out, that they may be so small as to require an immersion-lens for their demonstration. They may sometimes be made to appear by keeping the sputum in a moist chamber.

Their significance is not known. Leyden originally maintained that they caused the asthmatic attack by irritating the mucous membrane of the bronchioles, but their occurrence in so many other localities, as well as in nonasthmatic bronchial affections, weakens this theory. The presence of the crystals in nasal polypi, which are known to be at times responsible for asthmatic attacks, is very interesting.

In regard to the two patients in whose sputum the crystals were found, I may add that the man was not materially helped by treatment, but the boy, when last seen, was almost entirely well.

May 28, 1896.

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9. *Specimens from a case of typhoid fever with fatal relapse.*

Exhibited by Augustus A. Eshner, M.D.

A. R., a boy, aged fifteen years, who helped in the loading and driving of carts, came to the Out-patient Service of the Philadelphia

[1] Ladenburg. Quoted by Vaughan and Novy: "Ptomains and Leukomains," Philadelphia. 1891, 2d edition, p. 331.

[2] Cohn. Loc. cit.

Polyclinic on August 10, 1896, complaining of dizziness and diffuse backache, with pains in the feet and legs, which had developed in the sequence of excessive swimming during the very hot weather of the preceding week. There was some shortness of breath, with disturbance of sleep, impairment of appetite, and looseness of bowels. The tongue was rather heavily coated. There was little complaint of headache, and there had been no epistaxis. The pulmonary resonance was preserved, but the breathing was harsh, and sibilant râles were audible on auscultation. The temperature taken in the mouth was 103° F. There was some doubt whether the case was one of thermic fever, which was prevalent at the time, or typhoid fever, and the boy was advised to enter the wards of the hospital. He declined, but returned two days later, his previous symptoms having persisted, and he was then admitted. As the case developed, the clinical picture of typhoid fever unfolded itself, and the patient was bathed at a temperature of 68° F. whenever the temperature, taken in the rectum every three hours, rose to 102.8° F. or above, vigorous friction being employed and a stimulant being given in advance and sometimes also after the bath. He did exceedingly well for twenty days, requiring in all some nine baths. At the expiration of this time the temperature, morning and evening, had gradually fallen to the normal, and convalescence appeared about to set in. The diet was now enlarged by the addition of custard, junket, and other semisolid food. In the course of two days the temperature had again risen above the normal, and the patient passed though a relapse that terminated fatally by failure of respiration on the thirty-third day of observation. In this secondary attack twenty-seven baths were given. During the progress of the disease albumin appeared in the urine, and on one occasion it became necessary to use the catheter. Slight cough also developed and the sputum became blood-streaked.

Upon postmortem examination both lungs were found congested and edematous and containing numerous areas of lobular pneumonia. The right lung was universally bound by firm adhesions. The heart was small, the right ventricle relaxed, the left firmly contracted. The gall-bladder was distended with pale greenish mucoid fluid, and presented in its wall close to its junction with the cystic duct a firm fibrous nodule. The biliary passages were patulous. The spleen was enlarged and friable, measuring 15 x 9¾ x 5½ cm. Its convex surface displayed a circumscribed area of perisplenitis. The kidneys were rather pale and slightly swollen, but the cortex stripped readily. The mesenteric glands were greatly and universally enlarged and on section presented a somewhat fleshy appearance. The vermiform appendix was free and

7 cm. long. Its mucous membrane was the seat of hemorrhagic infiltration and of ulceration. The ileum contained many and large areas of ulceration in various stages of development and retrogression, while the colon contained numerous smaller areas.

This case does not demand extended comment, but a few remarks will not be superfluous. Throughout the first part of its course it appeared to be of mild type and pursued a favorable march. The relapse, on the contrary, soon showed itself to be more serious in nature, the nervous manifestations, previously insignificant, making themselves prominent. Clearly the primary attack had failed to confer immunity. In fact, the natural resistance seems to have been lowered. In how far the enlargement of the diet with defervescence influenced the occurrence of the relapse cannot be answered with any degree of positiveness. I am, however, not willing to withhold from that event some responsibility for the subsequent course of the case. While indiscretions in diet are not invariably followed by relapse or other complication, and while relapses sometimes result in spite of the greatest care with regard to the diet, still it should ever be borne in mind that too much caution cannot be exercised in the management of a disease whose uncertainty of course and termination constitute one of its most characteristic features. *September* 24, 1896.

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10. *A case of diabetes mellitus in a girl of thirteen years.*

Exhibited by David Riesman, M.D.

The young girl from whom the specimens I present this evening were obtained was under the care of Dr. L. Jurist only a few days. Sugar had been found in the urine, and, despite active treatment, coma developed, in which the patient died.

For the privilege of reporting the case I am indebted to Dr. Jurist. Of the history I only know that three years ago she had a fall. The following are the notes of the autopsy :

Body of a tall, fairly-developed girl ; pubic and mammary hair absent ; some abdominal distention ; a dirty fluid escapes from the nostrils.

Abdomen. The stomach is much distended and occupies a large part of the upper region of the abdomen. The appendix is normal and overhangs the pelvic brim. The diaphragm extends to the top of the fifth rib on the right side and the bottom of the fifth rib on the left.

Thorax. The pericardium is normal. The heart is rather large, but flabby. On puncture of the left ventricle with a needle, a little frothy, dark blood escapes, from which cover-glass preparations were made. The right cavities contain large currant-jelly clots; there is very little fluid on the left side. The muscular substance is quite soft. The anterior leaflet of the mitral valve has a spot of sclerosis. The intima of the aorta presents several miliary areas of atheroma.

The left pleural cavity contains some bloody fluid; a few firm adhesions are found at the apex of the right lung.

The lungs are congested at the base. At the right apex there are a few adhesions; some also between the lobes of this lung. The coal-dust is, perhaps, greater than one would expect in so young a person. There is no enlargement of the bronchial glands.

The spleen is small, the capsule wrinkled; there is a small supernumerary spleen.

The left kidney is somewhat enlarged; the capsule does not strip readily, and tears the cortex in a few places; the superficial veins are prominently marked. The relation of cortex and medulla is normal; the cortex is pale; the structural features are somewhat blurred. The right kidney is about the same, but the capsular adhesions are more marked. The suprarenals are normal.

The stomach is greatly enlarged; it was not opened.

The pancreas presents nothing abnormal to the naked eye.

The liver is of normal size and color, except for a number of irregular, yellowish-white areas scattered over the surface and extending for about 2 mm. into the substance of the organ; they resembled the necrotic foci seen in some of the infectious diseases. The substance of the liver is firm; the structural outlines are obscured.

The mesenteric glands are enlarged, but not caseous.

The thoracic and abdominal aorta present miliary patches and yellow lines of atheromatous change, particularly near the origin of the spinal arteries.

Pathologic diagnosis. Cloudy swelling of kidney and liver; slight interstitial inflammation of kidney; dilatation of stomach; hyperplasia of the mesenteric glands; atheroma of aorta; hemothorax; old pleurisy.

The blood was examined for glycogen and for fat; for the former by means of iodin-glycerin and iodin-gum-arabic solution, but no glycogen was found. This was to be expected, for the glycogen was probably converted into sugar soon after death. To determine the presence of fat the blood was fixed in Flemming's osmic-acid mixture and then

stained with eosin and hematoxylin. Examination shows the presence of fine black granules, which might be extraneous matter, yet, on a comparison with other blood-preparations fixed in the same manner, I do not find such granules. I am inclined to consider them fatty.

The blood in diabetes presents several interesting changes. As far as the number of corpuscles and the hemoglobin-percentage are concerned, departures from the normal are rare; in a few cases a light degree of anemia existed, and polycythemia in several others, especially in the stage of coma (Grawitz, *Klin. Pathologie des Blutes*, 1896, p. 158).

Bremer has described a peculiar reaction of the red corpuscles to stains. Normally the corpuscles are stained brownish-red by eosin; when the plasma contains an excess of sugar the red cells are either not stained at all by eosin or are tinged yellowish or greenish-yellow. The plasma is either unstained or has a pink hue.

Chemically, the blood undergoes various modifications. In the first place, there is a great increase in the quantity of sugar—normally, the blood contains (von Noorden, *Twentieth Century Practice*, vol. ii. p. 99) from 0.05 to 0.15 per cent. of sugar; in diabetes the amount has been found as high as 0.9 by Hoppe-Seyler (v. Jacksch, *Klin. Diagnostik*, 4th edit., 1896, p. 92), 0.23 and 0.48 by Seegen, and 0.57 by Pavy.

Glycogen, which, according to Gabritschewsky, is also found in normal blood, seems in diabetes to be increased. It appears in the form of granules staining brown with iodin, and occurs both within and outside of the white corpuscles.

Livierato, however, found but little extracellular glycogen in diabetes, and the researches of Czerny render it probable that the granules heretofore considered glycogen are in reality amyloid in nature (iodin, H_2SO_4, and methyl-violet reactions). I found no glycogen in the dead blood, but have given a possible reason for this.

Considerable attention has been bestowed upon the alkalinity of the blood, particularly since the view has gained ground that diabetic coma is the result of an intoxication by organic (fatty) acids. According to von Noorden, the alkalinity is normal as long as the patient enjoys fair health, but is decreased during the stage of oxybutyric-acid excretion and still more during coma.

Diabetic blood contains an excess of fat: a condition of lipemia exists, which, if very marked, may lead to lipuria, the appearance of fat in the urine. The causes of lipemia are not known. The presence of the fat can be demonstrated (Grawitz, *loc. cit.*, p. 160) by drawing the blood into a capillary pipet, and allowing the latter to lie horizontally for a time; the upper layer will appear creamy as if finely dusted with flour.

Cover-glass preparations can be stained with osmic acid: then, in order to test the nature of the stained granules, they may be dissolved in ether or xylol.

Another peculiarity of the blood is the reduction in the amount of the so-called "glycolytic ferment" of Lépine. The presence of a normal quantity of this prevents the accumulation of sugar in the blood: when there is a deficiency of the ferment the sugar is not destroyed, and the blood becomes overcharged with it. The ferment is believed to be furnished by the pancreas, and to be contained in the red corpuscles, and not in the serum. Recent investigations, however, throw a doubt on Lépine's theory.

Fatty acids (lipacidemia), particularly β-oxybutyric acid, have been found in diabetic blood by von Jaksch, Hougouneng, and others. Acetone may also be present.

A peculiar feature in the case here reported is the arteriosclerosis, which, in view of the patient's negative history, could only have been due to the diabetes. It is a well-known fact that diabetes produces a premature arterial degeneration, and in that way predisposes to gangrene, to intermittent claudication, apoplexy, etc. (Le Gendre, *Traité de Méd.*, t. i. p. 450).

The renal lesions in this case are probably best accounted for by ascribing them to the irritation of the sugar and of toxic agents in the blood.

Regarding the causes of diabetes, the history of this case contains but one fact of significance—the trauma. Kulz (*Handbuch der Kinderkrankheiten*, iii. 1, p. 269) in 111 cases found a history of trauma in six, of which four were cerebral injuries. In 108 cases collected by Wegeli (*Arch. f. Kinderheilkunde*, xix. 1 and 2), a history of injury was obtained in thirteen; as the cause was given or investigated in thirty-nine only, the disease was ascribed to injury in 33 per cent. of the cases. Ebstein has carefully investigated the question of the relation of trauma to diabetes, and has gathered fifty confirmatory cases, six of which occurred in his own practice. In half the cases the injury affected the head. Higgins and Ogden found glycosuria in twenty out of 211 cases of head-injuries. As a rule, the glycosuria disappears, but in a small proportion it becomes a permanent diabetes.

While all this evidence does not permit us to conclude that this case of diabetes resulted from the injury, particularly since the brain was not examined, we are justified in tentatively assigning some etiologic importance to it.

September 24, 1896.

11. *Urethral calculus and massive ventricular hemorrhage.*

Exhibited by T. S. Westcott, M.D.

The patient from whom these remarkable specimens came was a man about sixty years of age, who was brought to the Methodist Hospital by the patrol, with the brief history that he had fallen unconscious while at work digging for the foundations of a house. He was in deep coma, with contracted pupils, flushed face, stertorous respirations of 20 a minute, and complete muscular relaxation. Further examination showed the bladder greatly distended, reaching nearly to the umbilicus. On attempting to catheterize him, Dr. Crockett, of the resident staff, observed that the meatus was blocked by a foreign body, which could be felt as cylindric in shape, extending backward through the urethra for a distance of quite two inches. The penis was of large size, but the meatus unusually small and apparently retaining the foreign body by grasping it about a well-marked neck and allowing only a small cylindric knob to project through its lumen. Free incision of the meatus was required to allow removal of the body, which was delivered in several sections. It proved to be a calculous formation, apparently formed by slow deposit of urinary salts upon and behind several uric-acid concretions, which had evidently been retained behind the constricted meatus for a very long time. After its removal nearly two quarts of urine were evacuated from the bladder. Examination of this showed a small proportion of albumin, mucus, and pus.

Subsequent information from relatives elicited the following facts in the history: Had scarlatina in infancy; enlisted in army at the age of nineteen years, and served three years; after discharge suffered from bleeding hemorrhoids up to time of his death, often losing so much blood that he would become weak and faint. Has had difficulty in urination for many years, dating back to the war, and during the past two years he was known to be in the habit of using a long needle to open up the urethra when he wished to pass water. Has used whiskey and tobacco to excess. Ever since his discharge from the army he had mild delusions of persecution, carrying a pistol to protect himself against some unknown enemy. A doubtful history of syphilis contracted twenty to twenty-five years ago was given by his brother.

The case was then admitted to the medical ward. The condition was practically the same as already described. Respirations 24; temperature 100.8°; pulse 88, strong and bounding. In consultation with Dr.

Tunis, of the surgical staff, venesection was agreed upon, and a pint of blood removed from the left median cephalic vein, with relief of vascular tension. The bowels had been involuntarily opened while in the receiving ward. For further depletion a drop of croton-oil was placed upon the tongue. By 2 P.M. thirty-five ounces of urine, in addition to that removed earlier by catheter, had drained away. Toward evening, without change in the general condition, the temperature began to rise, and at 5.30 P.M. respiration became more labored and cerebral congestion more marked. Venesection was repeated by my resident, Dr. Deardorff, with temporary relief. The temperature, however, continued to rise, and when I saw him again, about 9 P.M., pulmonary edema had set in and the respirations had risen to 48, with a temperature of 105°, and pulse of 164. Atropin was given to sustain the respiration, and hypodermic stimulation by digitalis was added to the treatment.

Incidentally it occurred to me that in this terminal stage some relief of cerebral pressure might be effected by tapping the vertebral canal after the method proposed by Quincke, while at the same time some light might be thrown upon the diagnosis, though evidently at the eleventh hour. The needle of a large hypodermic syringe was accordingly inserted between the third and fourth lumbar vertebræ, about three-fourths of an inch from the median line. Fluid began to flow when the needle had reached a depth of 7 cm., and flowed steadily by rapid dropping until about six drams had been removed, when the needle was withdrawn. The fluid was deeply blood-stained. A culture of this fluid taken directly from the droppings yielded a negative result. The patient died about 1 A.M. of the following day, about sixteen hours after his seizure. Urine continued to flow in considerable quantity at intervals up to the time of his death.

The autopsy was made with great care and dexterity by Dr. A. E. Taylor, acting pathologist to the hospital. The notes are as follows:

Body of a muscular, middle-aged man. Rigor mortis well marked. Subcutaneous fat normal.

Peritoneum normal in appearance. Intestine distended, tympanitic; appendix points backward into pelvis, and is normal.

Diaphragm reaches to fourth rib on right side and eighth on left.

Spleen very congested, friable; capsule is sclerosed and thickened; left kidney fatty; capsule is thick and sclerotic; adheres closely. Upon section numerous cysts evacuated themselves of a purulent fluid containing much gravel. The substance of kidneys is fatty, fibrous, and presents cloudy swelling. Ureters are inflamed and thickened.

Bladder is small; walls extremely thick; mucous membrane is eroded and suppurating; bladder is full of purulent urine.

Pleura, left is free of adhesion; right pleural cavity is obliterated by fibrous adhesions which bind lung to chest-wall. Bases of both lungs are deeply congested, but there is no active formation of fibrin.

Pericardium sclerotic; fluid normal in quantity. Heart is moderately hypertrophied; weight, fifteen ounces; left ventricle extremely hypertrophied; wall one and one-third inches in thickness. Aortic valve normal, also mitral; right heart is dilated; the pulmonary valve normal; mitral admits two fingers. Aorta is sclerotic, but not dilated.

Prostate slightly enlarged.

Liver is enlarged, soft, and fatty. Stomach is normal in size; walls are very thin; mucous membrane is smooth and glistening, and presents the appearance of atrophy.

Brain: The skull is of average thickness; dura mater thickened, fibrous, and sclerosed; when detached small portions of brain can be seen on it. Vessels greatly congested; arteries at base very atheromatous. Upon section both lateral ventricles are filled with liquid blood, and there are large masses of clotted blood, particularly in the left. The left caudate nucleus is soft and broken. The origin of hemorrhage cannot be ascertained, but was doubtless from this softened area.

September 24, 1896.

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12. *Acute miliary tuberculosis.*

Exhibited by David Riesman, M.D.

The patient, J. E., was a tall, slender man of twenty-five years, a painter by occupation. The father is a drunkard; the mother died of dropsy, a sister of consumption. He has had scarlet fever, diphtheria, and possibly typhoid fever; never had lead-poisoning; he has used tobacco and alcohol moderately; is married two years, but has no children.

He was first seen July 9, 1896, and gave the following history: Four weeks previously he caught cold; had headache, pain in the chest, and some cough, with a thick expectoration, free from blood. After a week the cough and expectoration ceased, yet in spite of a good appetite he grew weaker, and was compelled to go to bed July 1st. Since then he has suffered from shortness of breath, a wheezing in the chest, and great prostration. He has no cough nor any pain; the bowels are regular.

Status præsens. On examination he was seen to be exceedingly emaciated and covered with a cold sweat; the tongue was bare and purplish in color; cyanosis was not present. There was no abdominal distention or tenderness; no spots; liver and spleen were not enlarged. The breathing was rapid, but not noisy; percussion-resonance was good; indeed, the note was hyperresonant, which I attributed to the thinness of the chest-wall. On palpation, a smooth, soft, to-and-fro friction fremitus was felt on the left side. On auscultation a chorus of small moist and dry râles, neither distinctly sibilant nor truly sonorous, could be heard, together with soft friction-sounds, all over the chest. Inspiration was a little harsh; expiration not prolonged nor harsh, except at the right apex. The heart-sounds were feeble; there was no murmur.

For two days the patient was rational, then a talkative delirium and carphologia developed; he also had an almost constant habit of sucking air through his teeth. The temperature varied from 100° to 101° F.; the pulse at first was 104, the respirations 42. Examination of the urine showed specific gravity 1022; color, dark amber, clear; considerable albumin; no sugar; no excess of indican; no diazo-reaction. Microscopically, crystals of magnesium phosphate were found.

A diagnosis of acute miliary tuberculosis of the lungs was made; the peculiar friction-rub on the left side was accounted for by assuming the existence of subpleural tubercles that rubbed against the parietal pleura. The pleura itself could not be involved, for both pain and effusion were absent.

The patient sank rapidly. On the 11th the pulse was 212, respirations 72; death took place on July 12th, three days after he was first seen and twelve days after he went to bed.

At the autopsy, made on the evening of the day of death, both lungs were found riddled with tubercles, which were practically all small, gray, miliary nodules; the lungs were crepitant everywhere except at the bases, where hypostatic congestion existed. The pleural cavities were dry; some adhesions were present at the left apex and between the lobes of the left lung. Beneath the pleura vast numbers of tubercles could be seen, and by passing the hand over the pleura could be readily felt. In the right apex an old calcified tubercle with a cheesy nucleus was found; it was two-fifths of an inch in diameter and one inch long.

Abdomen. The transverse colon was sigmoid in shape; beginning at the hepatic flexure, it passed transversely across the abdomen to a point on the left nipple-line, and then, curving upward into the left hypochondrium, touched the dome of the diaphragm. The intestines appeared normal. In the mesentery, at a point just behind the umbilicus, a large

tuberculous lymphatic gland, two inches long, one inch thick, on section showing a typical cheesy interior, was found.

On account of the presence of the wife, whose patience was vanishing, and the fear of the appearance of the drunken father on the scene, the autopsy from this point on had to be hastened. Rapid inspection of the liver and other abdominal viscera showed no tubercles. The brain was not examined; in all probability it was not affected.

Microscopic examination showed miliary tubercles with tubercle-bacilli in the lung; large numbers of miliary tubercles in the liver and caseous tuberculosis of the mesenteric gland. In the last I was unable to demonstrate the presence of tubercle-bacilli.

Remarks. There were two foci in this case, either of which might be considered the source of the miliary tuberculosis—the mesenteric gland and the calcified tubercle in the lung. I believe the former was the starting point of the disease—it was so much larger, and its situation with reference to the blood stream so much more favorable for the dissemination of the bacilli, that such a view impressed itself at once upon the mind. The involvement of the liver to a certain extent tends to corroborate this opinion, although the absence of tubercles in that organ would not of itself imply that the bacilli had not been conveyed along the portal circulation, for we know that abdominal tumors in the territory of that circulation can give metastasis to the lung without becoming localized in the liver. The pulmonary tubercle, finally, was obsolescent, partly calcified, and encapsulated, and had the appearance of the tubercles that are so frequently discovered accidentally in making autopsies on subjects otherwise not tuberculous.

No evidence was found of any spread along the thoracic duct.

From a clinical standpoint, if I might touch upon that in a few words, the case was interesting, first, from the absence of cough; secondly, from the absence of cyanosis; thirdly, from the comparative lowness of the temperature, and, fourthly, from the existence of the peculiar friction-rub, or, as I would term it, *subpleural friction*. In Fagge's *Practice* I find a reference to a paper by Jürgensen, who was the first to observe this phenomenon, and who considered it a pathognomonic sign of miliary tuberculosis of the lung. This case would seem to substantiate that view.

November 12, 1896.

13. *Senile gangrene of the left foot.*

Exhibited by H. A. Hare, M.D.

William J. M., aged sixty-three years, American; admitted to the Jefferson Hospital January 12, 1897; died February 9th.

Diagnosis. Senile gangrene (left foot).

Family history. Mother still living at eighty years and healthy. One sister died in infancy, one sister living and in good health, one brother died suddenly at fifty years from heart-trouble. Patient's maternal grandmother lost her mind some few years before her death.

Previous history. Fairly healthy as a lad, had the usual diseases of childhood. As a young man and as a middle-aged man led a healthy, temperate life. Never used distilled liquors as beverages, rarely malt-liquors, but has used tobacco freely. Has never had any specific trouble.

Present trouble. Five or six years ago began to notice a sense of uneasiness in bones of left foot; no acute pain, but a sensation of a tightly laced shoe, only noticed on walking, and would disappear after a short rest; condition steadily grew worse, patient being able to walk less distance, and requiring more frequent and longer resting spells. The pain, however, was not of an acute character, but rather that of sensitive bones distressed by a tight bandage.

In September the little toe of the left foot became inflamed and sensitive around the nail, and the doctor removed the nail under cocain-anesthesia. After this the pain became more acute and confined to foot and inner aspect of calf. At times has sensation of intense heat, at others of intense cold. There is some loss of tactile sense on outer surface of leg, beginning three or four inches below the knee. Some hyperesthesia of foot below ankle, increasing to diseased area. Slight impairment of heat-sensation in limb.

During stay in hospital the foot was kept dry and daily bathed with alcohol. The line of demarcation slowly formed transversely across the bases of the little and middle toes, which became dark and dry. Later the fourth toe became involved in the same process. Friday, February 5th, the dorsum of the foot showed irregular purplish mottlings. On the night of February 6th temperature rose and patient became much worse. By Sunday the foot had become black to the ankle-bend, but was dry. Sunday night the patient rapidly sank. At 10.30 intravenous injection of sodium-chlorid solution was given. Patient

immediately after had a severe convulsion, and sweating began and continued copiously during the remainder of the night. Patient kept alive only by the careful administration of stimulants. Became conscious at intervals, and by the next morning he had regained consciousness, and, while weak, was still able to talk to his family. He was conscious nearly the whole day Monday and the early part of the night. In the morning grew worse, became unconscious, and quietly expired at 9.10.

The urine, examined the second day before death, showed sugar, one-fourth of 1 per cent., by fermentation-test.

Postmortem notes. Incision in the median line shows the small amount of subcutaneous fat to be unusually yellow. Abdominal muscles friable and pale; liver 3 cm. below ribs in right mammillary line; colon normal; omentum normal, except for slight redness here and there. Abdominal organs in normal position; left pleural cavity, no adhesions; in right pleura, a few old adhesions at apex; the visceral layer of the pleura at this point is calcified, but there are no tubercles; 15 c.cm. of clear yellow liquid in pericardial sac; no evidence of pericarditis.

Left side of heart firmly contracted; right side flabby; right auricle distended with fluid blood and a large clot partially organized; antemortem and postmortem clots; small amount of blood in the right ventricle, also in the left auricle; left ventricle contains no blood; aortic and pulmonary valves are competent; mitral valves also; anterior aortic valve slightly thickened; some atheroma about opening of coronary arteries.

Right lung normal; left lung normal, except for pleural adhesions already named.

Spleen enlarged, substance light colored; capsule thickened in spots.

Both kidneys are movable; both suprarenals normal; right and left kidney normal, except for the rather thin cortex; capsule slightly adherent; parenchyma somewhat friable; lower part of ileum dark and black, as if gangrenous in patches, possibly postmortem congestion.

Pancreas normal but small. Liver a little smaller and paler than normal; somewhat friable. Bile-ducts patulous.

No abnormality of large vessels of thorax.

The foot was affected with moist gangrene from base of toes to instep; the toes were all involved in the process of dry gangrene. The leg was purple and mottled to the knee, but not edematous.

February 25, 1897.

14. *Septic abscess of foot.*

Presented by Oscar H. Allis, M.D.

Mrs. A., aged seventy-five years, married, has many grown-up children. Was attacked with erysipelas within the nose, which spread to her cheeks, ears, and forehead about the third or fourth day. A pain seized her in the left ankle, which had been sprained a year previous. The joint rapidly filled with pus and was opened by the family physician. I amputated above the ankle March 18, 1897. *March* 23, 1897.

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15. *Carcinoma of the extremities.*

Presented by G. A. Muehleck, M.D.

Prior to 1889, when Volkmann's[1] classical monograph on *Primary Carcinoma of the Extremities* appeared, this subject had received comparatively little attention from medical writers. Volkmann's critical analysis includes thirty-nine cases observed in his own clinic at Halle during the previous twenty years, all the cases published by others prior to 1889 and after 1860, and finally a considerable number reported to him from private sources—a total of 239 cases. To this number may be added fourteen cases reported by Schneider[2] from Bergmann's clinic, and finally twenty-six cases from Bruns's clinic, reported by Michaelis.[3]

Volkmann's classification of these neoplasms is now generally accepted by all writers on this subject. He divides them into three groups, as follows:

1. Those which originate on pathologic tissue, such as ulcers, fissures, fistulæ, scar-tissue; in short, from skin which has been the seat of long-continued irritation and injury. This is by far the largest group.
2. That group which comprises those cases which have their origin in warts and nævi, and, finally,
3. The group which grows from previously healthy skin.

[1] Volkmann. Sammlung klin. Vorträge, 1889, No. 334-35.
[2] Schneider. Inaug. Dissertation, Berlin, 1889.
[3] W. Michaelis. Beiträge zur klin. Chirurgie, 1891, Band vii. Heft 2, p. 421.

Classification of the cases according to sex shows a remarkable predominance of males over females in the ratio of about two to one. In the 205 cases of Volkmann in which the sex was mentioned, 140 were males and 65 females. Of Schneider's cases 9 were males and 5 females, and of Michaelis' cases 18 were males and 8 females, making a total of 167 males and only 78 females.

An explanation of this phenomenon may possibly be found in the greater liability to injuries and chronic irritation of the skin of the extremities in males, due to their more laborious occupations.

Such injuries and irritation of the skin being now generally accepted as an etiologic factor in the genesis of these growths, according to age we may classify them as follows:

Age.	Volkmann.	Schneider.	Michaelis.	Total.
20 to 30	5	1	0	6
30 " 40	24	0	1	25
40 " 50	52	2	5	59
50 " 60	56	9	7	72
60 " 70	38	1	10	49
70 " 80	22	0	3	25
80 " 90	1	1	0	2
	198	14	26	238

According to location the cases are classified as follows:

Upper Extremity.

	Volkmann.	Schneider.	Michaelis.	Total.
Arm	14	1	2	17
Forearm	18	0	2	20
Dorsal aspect of hand	56	2	7	65
Volar aspect of hand	1	1	1	3
	89	4	12	105

Lower Extremity.

	Volkmann.	Schneider.	Michaelis.	Total.
Thigh	23	2	1	26
Leg	75	4	12	91
Dorsal aspect of foot	19	3	0	22
Plantar aspect of foot	17	1	1	19
	134	10	14	158

These tables show that the lower extremities are far more frequently involved than the arm and forearm, the proportion being about three to two, the place of predilection being the leg, which is the seat of more than one-third of all carcinomata of the extremities. Following the classification of Volkmann as to etiology, Michaelis reports nineteen cases following chronic inflammation of the skin, two following warts and nævi, and five originating in previously healthy skin, showing that

three-quarters of all cases followed long-continued local irritation; and here, again, it is especially the chronic leg-ulcer which stands at the head of the list with almost one-half of the cases reported.

The appearances of these growths when developing at the site of an old leg-ulcer are quite characteristic and uniform, so that the diagnosis is always possible macroscopically. The previously flat granulations of the ulcer begin to proliferate and form cauliflower-excrescences with a decided tendency to ulcerate and break down. The cauliflower-excrescences may originate in small islands of epithelial cells still remaining at the bottom of the ulcer, in which case they usually grow rapidly and infiltrate the bottom of the ulcer. The already callous edges become more and more infiltrated, project over the bottom, and are more or less elevated above the surface of the surrounding skin, and finally the edges may be undermined and the growth penetrate the subjacent tissue, attack the periosteum, and expose the roughened and altered surface of the bone.

All these peculiarities are quite typical and readily permit a diagnosis by inspection alone. The neighboring lymphatic glands are almost always enlarged; but usually this is not due to cancerous infiltration, but is merely an inflammatory induration, such as is frequently found in chronic leg-ulcer without carcinoma.

In the majority of cases the swelling and induration disappear after thorough removal of the growth alone. Nevertheless, from a therapeutic stand-point it is important to remember that exceptions to this rule are by no means infrequent, and that true carcinomatous infiltrations occur.

Michaelis reports two cases in which compound comminuted fracture of the leg had preceded the leg-ulcer. In these cases extensive cicatrizations formed a favorable soil for the development of atypical epithelial growth, not to mention the possibility of epithelial cells becoming enclosed in the tissue-mass of the scar, and subsequently acting as germs for these growths.

In another case the growth developed at the site of an old leg-ulcer, which had been temporarily healed by Reverdin's skin-grafts. A similar case is reported by Volkmann (Case 70).

In a case reported by Michaelis, a cicatrix of the left index-finger of a peasant had existed for forty years, during which time, it is fair to assume, judging by the laborious occupations of these people, that the scar had been subject to frequent irritation until finally the epithelium assumed an atypical growth.

Carcinomata having their origin in warts and nævi are, according to Volkmann and Michaelis, also quite rare.

The majority of cases published under this category are to be classed

with the sarcomata. Volkmann reports twelve cases and Michaelis two cases of this group. Of these one-half were on the dorsal aspect of the hand, probably because of the frequency of warts in this locality.

It is of interest to note that all authors agree in attributing to these growths when developing from congenital warts a peculiar malignancy in spite of early and radical operative measures. The growth invariably recurred and proceeded to a fatal termination. Carcinomata of the third group—that is, those developing from previously healthy skin, are also quite rare. Among 183 of Volkmann's cases there were only 27, and among Michaelis' 26 cases there were only 5 belonging to this group; in all about one-sixth of all the cases observed. And here also the dorsal aspect of the hand seems to be the favorite seat for carcinomata of this group, twelve out of 27 of Volkmann's and 3 out of 5 of Michaelis' cases having their seat in this locality, and, as will be observed, this also holds good from previously healthy skin on the dorsal aspect of the thumb. It would be exceedingly difficult to explain this peculiar phenomenon. While it is true that the hand among the laboring classes is exposed to many injuries and its skin to almost constant irritation, yet it is a well-known fact that carcinomata of the palmar aspect of the hand are exceedingly rare, although it is the locality most exposed to injury and chronic irritation.

In the entire literature at my command only three such cases are recorded: one by Volkmann, one by Michaelis, and one by Schneider, from Bergmann's clinic.

Carcinomata of the extremities may, with few exceptions, as mentioned above, be classed with the most benign of malignant growths.

Thorough removal of the affected part, either by extirpation or amputation of the limb, has in the majority of cases assured against recurrence and effected a permanent cure, as shown by the following table:

	No recurrence.	Recurrence.	Total.
Volkmann	63	35	98
Schneider	8	4	12
Michaelis	10	3	13
	81	42	123

This includes all the cases which could be traced, the time of observation being from one and three-quarters to nine and a half years. Carcinomata of the extremities, therefore, admit of the most favorable prognosis of all carcinomata, excepting always that extremely malignant type originating from congenital warts. In this respect they must, therefore, be carefully distinguished from sarcomata of these localities, the extreme malignancy of which is the rule.

I am indebted to Dr. Robert Milligan, of the St. Agnes Hospital, for the history of the following case:

Wm. B., aged sixty-one years, white, laborer, German; no history of cancer or tuberculosis in family. No history of injury of any kind; patient's health is very good, eats well and sleeps well. About three months previous to admission a small furuncle appeared on the dorsal aspect of the thumb, which, he says, opened and discharged serosanguineous matter. At first there was but little pain and but slight enlargement. About the middle of January it began to grow rapidly, involving nearly the entire thumb, the hand becoming slightly edematous. Pain at this time was intense. After poulticing it he thought it became somewhat reduced in size. On first visit to clinic it resembled somewhat a syphilitic ulcer with slight seropurulent discharge. Pain was intense, radiating up the arm. Slight enlargement of axillary glands, but no involvement of the epitrochlear lymphatics was found. Amputation of hand by Dr. Wm. J. Taylor. *March* 25, 1897.

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16. *Multiple sarcomata in an infant.*

Exhibited by Augustus A. Eshner, M.D.

J. McC., a male child, aged two years and three months, was brought to the out-patient department of the Polyclinic Hospital on March 8, 1897, with the statement that he had been limping for about a month, the right foot turning inward. There had previously been no difficulty in walking, and no other abnormality had been noted. The child was extremely pallid and presented firm swellings in the temporoparietal, the temporofrontal, and the malar regions on the left side. The right knee was greatly enlarged and firm on palpation, and the thigh was almost cylindrical. The abdomen was tense and distended, and manipulation seemed to increase the child's distress. A mass could be distinguished on the right side which could not be separated from the liver. The child had suffered from no previous illness, and the first abnormal manifestation noted had been the pain at the right knee and the difficulty in walking, about a month before the child came under observation. There was no evidence of rhachitis. The parents were healthy, and the three other children in the family also were well. The latter had been born at term, without complication. During the eighth month of her pregnancy with the child the subject of this report the

mother had fallen in front of a trolley-car, but, although considerably frightened, she had escaped noteworthy injury.

The condition was looked upon as one of multiple sarcomata, and a fatal prognosis was given; but, not to ignore any possibility in the case, mercurial inunctions, with potassium iodid internally, were prescribed, and small doses of morphine given to make the child comfortable. Under this plan of treatment the little patient was kept alive for a month, the weakness and the pallor advancing progressively, and the several swellings slowly increasing in size. Upon *postmortem* examination, in addition to the pallor, the emaciation, the swellings mentioned, and several bedsores that had developed, the liver was found enlarged, extending three fingers'-breadth below the costal arch, far toward the left and pushing the lungs upward. These organs, with their pleural coverings, were otherwise normal. Heart and pericardium also were normal. The liver was normal, except for the presence of pale, apparently structureless areas surrounding the vessels. The left kidney was large, but appeared to be normal in texture. The right was constituted of a vast tumor adherent to all adjacent structures. On histologic examination, kindly made by Dr. Riesman, the growth proved to be a round-cell sarcoma. The superficial formations were not disturbed, out of respect for the wishes of the parents. It seems all but certain that the renal lesion was here the primary one, and the others secondary, although during life the possibility of primary involvement of one of the bones entering into the formation of the knee-joint, and probably of the femur, was taken into consideration.

The predisposition of the kidney to malignant disease differs radically from that of other organs in the fact that renal involvement is most common in the first months or years of life; it may even be apparent at birth. In the great majority of cases the new-growth is a sarcoma, with round or spindle or polymorphous cells; sometimes it presents the histologic structure of a rhabdomyoma, suggesting its origin in some developmental defect. The tumor is usually unilateral, one kidney suffering almost equally with the other. The two sexes also suffer almost equally. The disease is invariably fatal in the absence of surgical intervention, of which, it must be confessed, the results have not been very encouraging. If, however, extensive metastasis has not taken place, the child should be given at least the small chance of escape that timely operation affords. *June* 10, 1897.

IX. TECHNIC.

1. *On the preparation of macroscopic eye-specimens.*

Exhibited by William Campbell Posey, M.D.

It is my wish to exhibit to you this evening a series of macroscopic specimens of eyes which I have prepared during the past winter in the laboratory of the State Hospital for the Insane at Norristown. I do this with a certain reluctance, as I fear that most of you will find but little pleasure in listening to a description of a group of diseases which are, perhaps, unknown to you as general pathologists; but I trust that the beauty of the specimens may repay you for viewing them. The method of preparing them was taught me by Mr. Treacher Collins, of London, curator of the museum in the Moorfields Eye Hospital, and is practically the same as that which was introduced by Mr. Priestley Smith some years ago.

The glass jars which I employ have been made for me by the Fox Optical Company, and may be obtained at a reasonable rate. They consist, as you see, of a simple glass jar which is capped with a plano-convex lens. This small disk of opaque glass is used to seal the jar, and forms an excellent background for the specimen. A convenient receptacle for the lens is offered by this block of wood. (Demonstration.)

The preparation of the eye for mounting is briefly as follows: The entire globe is hardened in Müller's fluid for at least three weeks. It is then placed in a freezing mixture of salt and ice, care being taken to envelop the eye in oiled silk to prevent any of the fluid from the freezing mixture coming in contact with it. After the globe is thoroughly hardened it is divided by a bold stroke of a knife, that form of knife used in sectioning the brain being the best adapted for this purpose. One-half of the eye is reserved for microscopic examination, whilst that which is to be mounted macroscopically is placed in water to thaw out and to rid it of the excess of Müller's fluid. To remove the stain caused by the fluid more effectually, the section of eye is then placed in a 5 per cent. solution of chloral hydrate, care being taken to change the solution daily until the section is no longer discolored. As the globe would shrink were it placed in the mounting fluid at this

stage, it is first subjected to solutions of glycerin and water of different strengths. The eye is then ready for mounting.

The preparation of the medium used for this purpose is the only difficult stage in the entire process. It is quite a painstaking task to obtain a clear solution, and one frequently consumes three or four hours in the process, to be rewarded by a product which is too cloudy for use. The solution is best prepared as follows: One ounce (about twelve to fourteen strips) of the best French gelatin (Coignet & Company, Paris) is cut into small bits by a pair of scissors, eight ounces of water are then added to the gelatin, and the mass set aside in a beaker to swell. The whites of two eggs are then taken, and better to clear the mixture later on, the shells of the eggs are cut into fine pieces and thoroughly mixed with the albumin. The gelatin is now melted with a gentle heat, a few drops of carbolic acid being added as an antiseptic. As soon as it begins to boil the albumin is added, and the whole mass stirred with a glass rod. As the mixture is very readily scorched, constant attention must be given it until the fluid is quite clear below and a thick scum has gathered upon the top. The mixture is now filtered through flannel, or preferably through a steam filter, and the product, which should be quite clear, is collected in a jar which has been made perfectly clean. Eight ounces of glycerin are then added. After the glycerin and jelly have been thoroughly mixed, one of the glass cells is two-thirds filled with the solution, and the section of eye floated upon it. The globe is then seized at the equator with a pair of forceps and gently rolled over in such a way that no air-bubbles are permitted to get into its concavity. Should any bubbles, however, get in, they can be readily removed by sucking them out through a glass tube. A hand-mirror is now placed under the specimen and a good view is obtained of the position of the eye and of any air-bubbles which may have escaped detection from above. The globe is then kept from rising in the fluid by placing over it a bit of card-board, perforated by a pin.

The jar should never be entirely filled with the solution, for it is always better to leave an air-space between it and the opaque disk which is cemented on after the jelly has thoroughly set.

I shall hand around these specimens that you may the more readily study them. You will see the globe somewhat magnified, the effect of the planoconvex lens.

Specimen 1. Normal eye. This eye was removed from an inmate of the Norristown Hospital shortly after death, and shows very beautifully the structure of the normal eye. The relative thickness of the sclerotic

and cornea, and the topographical relation of the anterior and posterior chambers of the eye, can be readily studied. The optic nerve has been cut directly in half, so that the physiological excavation in its head is plainly visible. The retinal vessels are also seen coursing over that delicate sheet, and its pigment-layer can be well seen as it continues forward to form the posterior layer of the iris.

Specimen 2. Hemorrhage into the ciliary body. This eye was presented to me by Mr. Collins. It was enucleated in the Moorfields clinic on account of pain. It shows very beautifully a hemorrhage that has been localized in the ciliary body. The angles of the anterior chamber are patulous, and the optic nerve-head is not excavated, showing that if a glaucomatous state had necessitated the removal of the eye the increase in intra-ocular tension could not have been of long standing. The vitreous appears shrunken and has contracted into a flocculent mass directly posterior to the lens. The numerous rucks in the retina are of postmortem origin. This specimen has been in my possession four years and exemplifies very well the durability of this method of mounting, for the jelly is just as clear and as firm as the day upon which it was poured into the cell.

Specimen 3. Chronic glaucoma. This eye was given me for mounting by Dr. Risley, and exhibits in a very striking manner the essential pathologic features of that disease. In the first place, the globe is seen to be rather short, and was probably a hypermetropic eye. Again, the lens seems to be relatively large, and the space between the margin of the lens and ciliary body appears to the naked eye to be very much compromised. The angles of the anterior chamber are completely blocked; the pupil is widely dilated. The optic nerve-head is cupped in quite a different manner from that in the normal eye, for if you will look closely you will see that the excavation comprises the entire head of the nerve, whereas in the normal eye the cup was confined to its central portion about the entrance of the retinal vessels.

Specimen 4. Myopia and anterior polar cataract. This eye was enucleated by Dr. Zimmerman. I show you this specimen next that you may contrast it with the preceding, for one is at once struck with the difference in length of the two eyes. I should think from the length of this globe that there must have been a myopia of over 20 D. You will also observe that the eye gives the impression of overstretching. Its walls are much thinned, the sclerotic being very thin in places, and in the posterior pole of the eye there are large, whitish-looking areas which indicate patches of choroidal absorption, the sclerotic being visible in the bottom of the area. As often happens in these cases, the

vitreous is much shrunken and the retina is seen to be detached below. If you will look at the cornea you will find it to be opaque in its central portion. This opacity is directly continuous with a whitish mass which is in intimate association with the anterior capsule of the lens. There has been a primary ulcer of the cornea in this eye which has so thinned that membrane that it has given way to the intra-ocular tension and has perforated. The lens has been pushed forward at the time of the escape of the aqueous humor, and as a consequence of the impaired nutrition to which the cells lining the anterior capsule were subjected, this prominence, or what is known as anterior polar cataract, has formed.

The three other eyes which I will now exhibit were enucleated in our clinic at Wills Eye Hospital by my chief, Dr. Oliver.

Specimen 5. Intra-ocular growth. This is a most unusual specimen, representing very beautifully a neoplasm of the anterior segment of the eye. The case is now being carefully studied, and will be reported in full elsewhere. I may state, however, that the patient was referred from the country for enucleation with a diagnosis of iridocyclitis, although there had been some evidence of intra-ocular growth. Upon viewing this specimen one is instantly struck by the contrast in color which is presented by the different parts of the eye. This brownish-red mass I take to be the neoplasm, whilst the yellowish mass which appears to fill in the interstices is probably purulent. A similar case was reported in Graefe's *Archiv für Ophthalmologie* last year, and throws considerable light upon the one under discussion. I am inclined myself, from the microscopic examination, to consider the growth to be gliomatous in nature, as it consists of cells which are distinctly neurogliar in type.

Specimen 6. Iridocyclitis. This eye was enucleated on account of pain. This symptom was occasioned by the development of secondary glaucoma as a consequence of a stasis in the intra-ocular circulation caused by a blocking up of the anterior chamber. The fluid in the posterior chamber had driven the iris forward to such an extent that the membrane was in contact with the cornea. The iris, being bound down to the lens at its pupillary edge by firm adhesions, and being attached to the ciliary body peripherally, has an arched appearance, and deserves the appellation of *iris bombe*. The vitreous humor has shrunken, and in its place broad bands of connective tissue are seen which traverse that humor. The lens is encapsulated in a mass of newly-formed inflammatory material. By throwing the light into the deeper portion of the specimen by means of this strong convex lens which I hold in

my hand, one is able to see very beautifully a number of fine filamentous processes which are given off from the large masses of connective tissue. The retina is completely detached upon one side, probably as a result of the contraction of these bands. A large hemorrhage is seen to the nasal side of the eye occupying the internal retinal layers.

Specimen 7. Perforating wound of the eye. This last specimen shows very well a frequent result of this class of injuries. A bit of steel had perforated the cornea and the lens of this eye, and had been successfully removed; notwithstanding this, however, symptoms of sympathetic inflammation supervened in the other eye, and the offending and exciting eye was removed. The configuration of the eye appears perfect. There is a broad band of newly-formed tissue which stretches across between the two chambers of the eye. This is composed of lenticular débris, newly-formed capsular and connective tissue, and the iris which is glued into the mass. I have examined the optic nerve of this eye microscopically, but find no evidence of inflammatory change in it, which is additional evidence that the disease does not always affect the sympathizing eye by an inflammation which is propagated along that nerve. *December* 12, 1895.

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2. *Röntgen's discovery as applied to medicine.*[1]

Read by Henry W. Cattell, M.D.

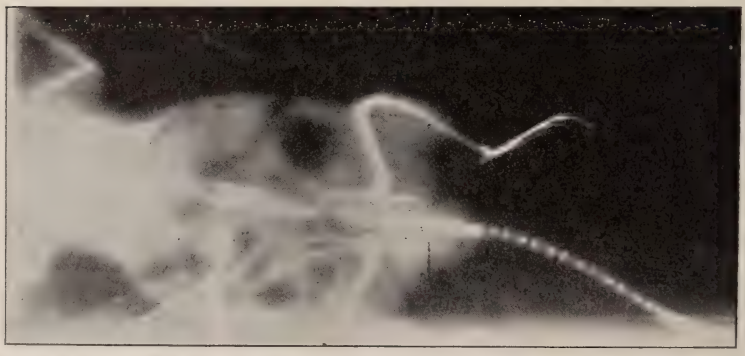
The weird and wonderful discovery of a peculiar form of energy by William Konrad Röntgen, Professor of Physics in the University of Würzburg, marks a new era in practical medicine, for we are now able by this process to produce pictures of bones and certain other substances, such as glass, bullets, and pins, when covered over by the soft tissues of the living body. The imagination will at once suggest the manifold uses to which such pictures can be put, and, according to the accounts in the daily papers and from our own experiments, results have already been obtained but little expected even a few months ago.

[1] At this meeting of the Society abstracts were read from Röntgen's original paper entitled "A New Form of Radiation," the entire article having been kindly translated for the writer by Dr. William G. Spiller. The apparatus for making Röntgen-pictures was demonstrated with a number of skiagraphs taken in Philadelphia. A more elaborate article on this subject was published later by the writer in the July 1896 number of the International Clinics. Owing to my election in May as Coroner's Physician, I was obliged, very reluctantly, to give up work in this fascinating field of original research. Our thanks are extended to the International Medical Magazine Co. for the loan of Figs. 1 and 2, and to Lea Brothers & Co. for Figs. 3 to 6.

Röntgen has found that the electric current passed through a Ruhmkorff coil, and thence to a Crookes vacuum-tube, develops from the cathode an invisible form of energy having the double property of penetrating certain ordinarily opaque substances and of affecting an ordinary photographic plate. Flesh, for instance, is relatively translucent to these rays, and bone, metal, glass, and graphite are comparatively impervious. On this difference in penetrability depend the effects which are permanently recorded by ordinary photographic plates. It will be convenient to coin the word skiagraph (*σκία*, a shadow), or skotograph (*σκοτός*, darkness), to characterize pictures produced by the x-rays, and to indicate at once their relationship to and their distinction from photographs (*φῶς*, light).

The method is very simple, in fact, so easy of accomplishment that, the process being known, one wonders, as is often the case in similar instances, why it was not discovered before. The technic is as follows: An ordinary photographic plate is introduced into a wooden plateholder,[1] which, in turn, is placed upon a firm table. The hand or other object to be skiagraphed is now rested upon the plateholder, and a Crookes tube[2] is arranged from three to four inches above the object,

FIG. 1.

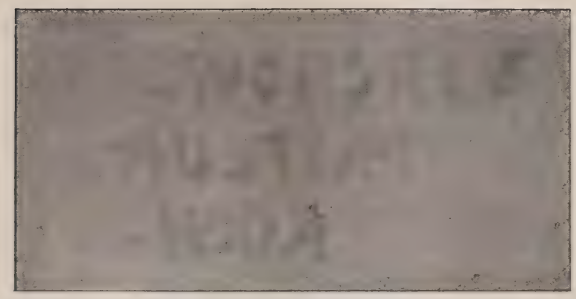


Portion of a mouse. A positive was prepared, and the half-tone cut has been made from this.

so that the x or cathodic rays will fall perpendicularly upon the object. An ordinary Ruhmkorff coil and several storage-cells are sufficient to supply the electric current, though a higher frequency current is better, as the time of the exposure can thus be materially lessened. In the Physical Laboratory of the University of Pennsylvania, where, through the kindness of Professor Arthur W. Goodspeed, who was the first in

[1] Or the plate may be wrapped in several thicknesses of orange or black paper.
[2] See any work on physics or an encyclopedia.

FIG. 2.



Words written with chromate of lead oil-paint on an ordinary visiting-card, which was placed on eight thicknesses of orange photographic paper and then skiagraphed. The name printed on the card in printer's ink does not appear, while the other words are of course reversed.

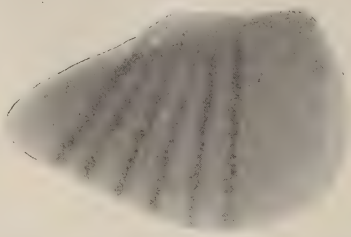
Philadelphia to test this new discovery, experiments have been carried on by the writer, the length of exposure has been an hour or more,

FIG. 3.

Small intestine containing cent, lead-pencil, and gallstone: the latter, being organic matter, is very indistiuct.

while, in other places, with more perfect apparatus, even four and a half minutes have sufficed. After the exposure the sensitized plate is developed by any of the ordinary methods, and prints can then of course be prepared from the negative. In the accompanying illustrations (Figs. 1, 2, 3, 4, 5, and 6) may be seen several untouched half-tone reproductions of skiagraphs taken from among the objects first employed by the writer.[1]

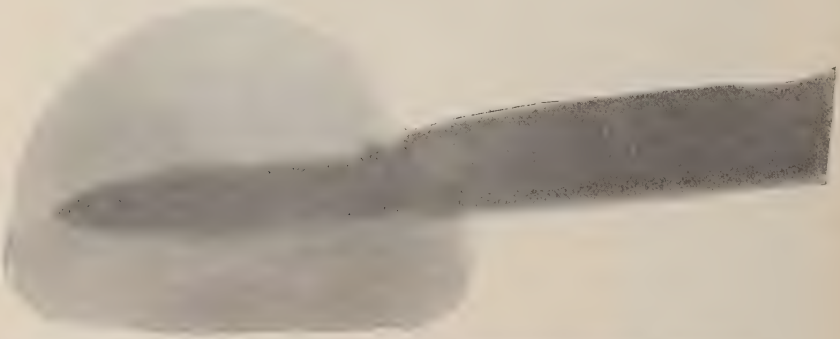
FIG. 4.



Six pins in center of section of cirrhotic liver one centimeter thick.

It would be interesting to see a skiagraph of the entire human body, though difficulty will be met with in those places where the shadow of one bone will be cast upon another, the bones being at different distances and planes from the sensitized plate. Edison is trying to overcome this by fixing numerous places from which the x-rays will emanate. The

FIG. 5.



Blade of penknife covered by two and three-quarters centimeters of lung tissue.

superimposing of the skiagraph upon the photograph, the latter possibly in color, would certainly make a novel and instructive picture, especially if an anaglyphic effect could be produced at the same time.

[1] As the work represents some of the first original experiments performed by any one on medical subjects, the article is printed just as delivered (see Medical News, February 15, 1896, and International Medical Magazine, February, 1896).

I have seen the fresh specimens from two postmortems where an unsuspected jack-stone was lodged in the larynx. As the tissues here are capable of being skiagraphed by the apparatus already at our command, the laryngologist has a new instrument which he can use for such purposes. It is stated that stone in the kidney has already been detected, and the opacity of glass has led to the finding of small pieces adherent to bone after accident. The similar opacity of lead may render the probe useless in gunshot-wounds, except in rare cases. A new means of distinguishing luxations from fractures is now added to the long list at our command. Obstetricians will readily perceive the immense value of being able to *see* the fetus *in utero* after ossification of its bones has occurred. The representation of deformed pelvis in the living subject, of spondylolisthesis, of calcareous infiltration of various

FIG. 6.

Anchylosis of terminal phalanges. Enlargement of second phalanx. (Arthritis in living subject.)

parts, such as arteries, and of exostosis—all this opens up a tempting and promising field for future research. It is expected that in the preparation of pictures from morbid growths the new discovery will be of some use, as the intensity of the shadow due to the different thicknesses of the tissues through which the rays will act will be shown up in the plate. Thus an absent or horseshoe kidney, an abscess, or a tumor might possibly be discovered. Perhaps it can be told when a hip-joint has become fixed in coxalgia. It will be comparatively easy to obtain pictures of acromegaly, osteitis deformans, gouty deformities, rheumatoid arthritis, etc. Especial attention will be given to the differentiation between organic and inorganic substances, and between those tissues in the human body which react to the rays and those which do not.

The easy application of Röntgen's method of taking a picture renders its use at once possible in hospitals. The entire cost of such an apparatus need not exceed $50. Doubtless the scientific and commercial ingenuity now being concentrated on this process will soon produce outfits at once simple, convenient, and portable, and it is safe to say that

most practitioners will then provide themselves with the means of seeing into the mysterious recessess of the body now accessible only by means of the knife.

Discussion.—Dr. Ernest Laplace said that, with reference to the work in which he was especially interested, the brain, he failed to see how, the skull being less permeable to the rays than the softer tissues, the brain could be photographed, and hence a bullet located in it. Aside from this, as far as its aid to surgery was concerned, it was almost unbounded, and no one could begin to say precisely where it might lead. Skiagraphy was as yet in its infancy, and was scarcely developed to any practical extent. All would join in admiration of the genius of the man who had brought the method to light.

Dr. William Thomson asked if the apparatus exhibited would be at all sufficient to make experiments, or whether the two cells and the induction-coil would require so long an exposure as to make it impracticable. Whether a more powerful induction-coil and the Edison current would be all that was necessary.

He thought the use of the extreme sensibility to the bromid of silver would be an advantage. It seemed to him that it might be possible to see the shadows without going to the length of time to develop them. The surgeon might be able to carry on his observations with a fluorescent screen.

Dr. Judson Daland had been very much interested in the thought of a possible influence that this light might have on micro-organisms. He did not know whether there had been any work done in this direction, but thought it would be a very interesting and, at the same time, a very simple problem.

Dr. A. C. Abbott believed Mr. Edison was endeavoring to determine the germicidal effect of the rays. Dr. Cattell had already given a number of suggestions as to what might be done with this apparatus. Whether the light would have any effect upon living animal tissue in its application to such tissues for the purpose of destroying bacteria, that is to say, whether the tissues would also be destroyed or not, was a question. It was quite possible that the rays might have a destructive effect upon bacteria and not upon the tissues. This was a supposition within the bounds of possibility. A short time ago we should have been surprised if we had been told that the rays of the sun were destructive to bacteria, whereas it was a perfectly well-known fact now. Men were also surprised to learn a few years ago, from Germany, that not only the direct rays of the sun, but diffuse daylight killed bacteria.

He wished to ask one question, and that was if any efforts had been made to concentrate the rays in a way that would be analogous to the concentration of the ordinary diffuse light, as in microphotography. It could not be accomplished in the same manner because of the impenetrability of glass, and something else than glass would have to be used. It would be very interesting if these rays could be condensed by opaque lenses. He desired to thank Dr. Cattell for the pleasure he had had in hearing his paper.

Dr. G. G. Davis thought the apparatus was a very elementary one, and before it could be of much practical use considerable advances would have to be made in the method of its application. The two cells were simple enough, and the amount of electricity which they furnished was easily handled; but, as he understood it, the apparatus would require so long to give an image as to make it inapplicable. In living subjects it would be impossible to hold them quiet a sufficient length of time to get any image. When it came to getting rays of such a strength as to be of practical use, he understood that they had to use a street-current. Mr. Edison's experiments might lead to something in the way of simplification of the tubes and of the coil which was used at present. The photograph of the hand which Dr. Cattell presented showed that the strength of current used for that photograph was apparently only sufficient to penetrate the thickness of the hand. This would prevent it showing anything at a greater depth than the fingers of the hand. He thought it would have to be worked up more extensively before it could be of any practical value.

Dr. Cattell, replying to Dr. Thomson's questions, said the exposure would have to be eight or ten hours in cases where the two cells and the induction-coil presented were used, but the Edison current would make this all right. There had been several announcements from Italy that experiments with the fluorescent screen had been made.

In reply to Dr. Abbott, Dr. Cattell said that concentration of the rays had already been attempted by placing a bell-jar over the tubes. There was in all probability a way of focusing the rays in such a manner that the shape would appear more distinct and the time of exposure would be lessened. Substances had already been suggested out of which a lens might be made. It must be remembered that the experiments of Röntgen were conducted before it was found that the rays did not refract. They would pass through the object without being turned aside, and thus the difficulty of bringing them to a focus could be understood. The rays of light would pass through ordinary slate, so that this was a substance which would not be of advantage. It seemed

to him that one could control the light in such a manner that it would be more sharply defined. It might be possible to make a picture of the entire body. When one looked back upon the first attempt that was made, and considered the length of time, it was remarkable to think that it might only require three or four minutes to take a picture. The apparatus which he showed had not been used for making pictures. It merely illustrated the technic of the process. The technic, he believed, would be markedly improved. Certain places would be found to be more sensitive than others, the difficulty with regard to the tubes would be overcome, and a large number would be made. He had gone very carefully over the things necessary in getting the apparatus together, and the expense connected therewith, and had found that the ordinary instruments, including everything necessary for the taking of a picture in three minutes, would not cost more than $50 or $75, depending upon the expense of the coil. This would bring it within the reach of hospitals and physicians. *February* 13, 1896.

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3. *A new postmortem case.*

Exhibited by Judson Daland, M.D.

The making of autopsies is a subject in which we are all interested, and it occurred to me that it might be of advantage to go over a few points in reference to the subject, although I am quite clear that it is an old one to most of you. In making autopsies in private I find it useful to place the case in an ordinary student's bag made of flannel, as it attracts less attention. After the case is removed you can use the bag to remove any specimens which you may desire to carry away, and no one would notice the substitution. The case itself is of moderate size, and has been in actual use for about ten years. Most of the advantages it possesses have been in actual practice. One advantage is that it can be very readily cleansed. While we have all been compelled to make autopsies with a simple knife, yet we prefer everything that is necessary for our use. This probe that I show I have found very useful, especially in the gall-ducts, as it facilitates their examination, and the little syringe I find of value in collecting small quantities of liquid, as liquids are often so placed that it is inconvenient to secure them. The ordinary forceps is used, and the employment of the blow-pipe is of importance, more particularly in working about the pleura to locate some point of rupture. With

the grooved director you are all familiar. One small knife is all that is necessary, and the knife which is used generally is the one which is accurately modelled after the one employed by Virchow. The apparent roughness of the handle is a distinct advantage, as it enables you to get a more certain grip. The use of the tenaculum is not often called for. This instrument is intended for carrying sutures about the intestines or any part you may wish to ligate. The knife I now show is constructed like the preceding, but is smaller and is unusually strong. You will observe a button on the back for the index-finger. When applied to the thorax it cuts through the cartilages. You can cut them all without injury to the thoracic structures, and it saves the edge of a cutting knife. The knives should be made particularly strong, and you want a big handle in order to apply sufficient force. Opening the skull is rather tedious, and it has seemed to me that this sort of saw is the best. It is somewhat rounded, and in using it it seemed to me most convenient to pass it backward and forward without using any downward pressure, and allow the points to go gradually through the bone. In working around corners it has a certain amount of flexibility. The use of the enterotome is known to you all. Another use is cutting along the heart into the aorta or the pulmonary artery, and this instrument answers very well for that purpose. A small pair of scissors with a small bell-like extension of one point is useful in following up small bronchioles. Sometimes you desire to make some estimate regarding the weight of organs, and this little scale will do this approximately. It occupies very little space and gives very good results. In making autopsies in private it is rather desirable that the relatives should not hear loud noises, and in attempting to lessen the sound you place this cap over the head of the hammer, which considerably lessens the noise. The hook at the end should be blunt, not sharp, otherwise the hand will be lacerated. The instrument for cutting the ribs has one point that I wish to mention, and that is that the blade will not turn inward. The hand is frequently caught. Many of us have had difficulty in removing the brain after dividing everything else, and we oftentimes desire to divide the spinal cord as low down as possible. I have a special knife for this purpose which is one and one-quarter inches long. You can go into the cervical region and divide the vertebral arteries and the spinal cord as low down as possible, without opening the spinal canal. This, I think, is extremely useful. The ordinary brain-knife you will find of use. In one corner of the case is an ordinary measure, which you may need to use in measuring various organs, and in another corner is a small jar of grease. It is gen-

erally desirable to anoint one's hands as thoroughly as possible. It is often impossible to get a piece of string or thread in the house, and I find it best to have a little spool of cotton in the case, as it occupies a very little space. The needle that seems to me best is one of the large ones that is slightly curved, with three edges. With this thread one can, I think, sew up an incision as rapidly as possible. The ordinary probe, of course, is useful, and I think the little probe of the otologist is the best. The little ivory measure that Hawksley often gives is extremely useful in measuring organs in situ. *March* 26, 1896.

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4. *Preparation of frozen sections by means of methyl and ethyl chlorid.*

By Henry W. Cattell, M.D.

In September, 1895, while watching Dr. John B. Deaver perform a minor surgical operation with ethyl chlorid, the thought occurred to me that this reagent might profitably be employed for preparing frozen sections for histologic purposes. The results thus far obtained have been exceedingly satisfactory, and, while the method is somewhat expensive, no accessory apparatus is required for the microtome.

Hamilton's method of preparing the tissues for freezing gives good results (*Text-book of Pathology*, by D. J. Hamilton, vol. i., p. 58). Another way of getting the tissues ready is that recently advised by J. Orth (*Berl. klin. Woch.*, 1896, No. 13): 100 parts of Müller's fluid are mixed, when wanted, with 10 parts of formol. Small pieces of the tissue under examination are fixed and hardened in this solution in the incubator for three hours. At the end of this time they are removed and thoroughly washed, and alcohol is gradually added until they are placed in 95 per cent. alcohol. This latter reagent must, of course, be removed before the tissue is frozen. If desired, after washing the specimen may be at once transferred to the solution of acacia and sugar, and frozen. Or, as suggested by H. Plenge (*Virchow's Archiv*, vol. cxliv. p. 409) the pieces may be placed in a 4 per cent. formaldehyd-solution for a quarter of an hour and then frozen in the same solution.

When the tissue has been prepared in some such manner, or even when perfectly fresh, it is placed with some excess of gum-acacia fluid upon the specimen-holder of the microtome, and a small stream of ethyl chlorid, methyl chlorid, or anestile (a mixture of these two reagents) is played from above directly upon the specimen. The tube

containing the ethyl chlorid is held about a foot from the specimen, and moved from place to place until the specimen is firmly attached to its base of support, and the upper portion is coated with a few crystals of ice. These crystals are extremely small and delicate, and therefore do not injure the tissue so markedly as in some other of the freezing methods. The specimen is readily frozen in from thirty seconds to a minute. Sections are then cut and placed in water or 50 per cent. alcohol, and mounted in the usual way. Excellent stained preparations may be prepared in fifteen minutes or less from the time that the tissue is removed from the body. *December* 10, 1896.

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